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HISTOPATHOLOGY OF THE TEETH

AND

THEIR SURROUNDING STRUCTURES

BY

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PREFACE TO THE SECOND EDITION.

THIS book has been enlarged, rearranged, and completely rewritten for the second edition. The subjects have been grouped somewhat differently, as suggested in the Report of the Curriculum Survey Committee of the American Association of Dental Schools. More than before, an attempt has been made to omit controversial and, to the tyro, rather inconsequential details, and as far as possible to offer an unbiased survey of present-day knowledge of the various subjects.

The chapter on the Histology and Physiology of the Dental Tissues has been omitted because its contents were since enlarged and incorporated in the author's *Dental Histology and Comparative Dental Anatomy*. The chapter on Histological Technique has been dropped since it is outside the sphere of interest of the dental student and practitioner; those interested in the preparation of specimens of the type illustrated in this book are referred to the article, "A Technique for the Preparation of Histologic Sections Through Teeth and Jaws for Teaching and Research" in the *Journal of Dental Research*, June, 1937.

Among the chapters or subjects included in this new edition are: anomalies in shape and number of teeth; physiology and pathology of tooth calcification; Hutchinson's teeth and mottled enamel; erosion; bacteriology of caries, pulpitis, and periodontitis; focal infection; pulp amputation; healing of extraction wounds; Vincent's infection; discolorations and stains; some of the diseases of the oral mucosa; tumors of the oral cavity; and differential diagnosis of toothache and of cysts of the jaws. The number of illustrations has been increased from 385 to 438.

The bibliography is confined to fairly recent publications that can be easily secured; most of the articles cited contain all the needed references to earlier publications on the particular subject.

The author is greatly indebted to The Foundation for Dental Research of the Chicago College of Dental Surgery under whose auspices nearly all our recent research work has been carried out, and to the Chicago College of Dental Surgery, Dental School of Loyola University, from whose Research Department most of the findings reported in the first edition originated. Most of the

new illustrations have been taken from material and publications of The Foundation and its workers. Drs. Kanner, Tunnicliff, and Willman have read parts of the manuscript and have contributed helpful criticism and advice. Miss Maurine Willman has been instrumental in getting the manuscript in shape; Mr. P. M. Orlopp had charge of the photographic work connected with the illustrations. Lea & Febiger have been of great assistance by the usual excellent coöperation of their manufacturing department.

R. K.

CHICAGO, ILL.

PREFACE TO THE FIRST EDITION.

THE rapid progress in microscopic dental research during the last few years has brought forth so many new and fundamental facts that a description of these recent findings together with the resulting new conceptions of old problems needs no justification.

Previous to the last decade, dental research made use of only three types of specimens for microscopic studies, namely, extracted human teeth, animal jaws, and dried human bone specimens. By means of such material much valuable and fundamental knowledge of a great many subjects in normal and abnormal dental histology was gained. While a study of dental caries and a study of the histology and pathology of enamel, dentin, and pulp could be made satisfactorily from ground or decalcified sections of extracted human teeth, normal or pathological conditions lying beyond the tooth proper presented an entirely different problem. Here the study of extracted teeth could not give the desired information. The older investigators, realizing this limitation, took refuge in the study of animal tissues, and microscopic descriptions of the hard and soft tissues investing the tooth were obtained largely from specimens of sheep, dogs, and other animals. But it remained an open question to what extent the periodontal tissues of these animals compared with the corresponding structures in man. Thus there was evident the need for a new type of investigation, namely, the study of human teeth in connection with their investing hard and soft tissues under normal and pathological conditions.

Some problems discussed in this book, such as the histology of the epithelial attachment and the gingival crevice or the influence of normal functional conditions upon teeth and surrounding structures, belong to the field of physiology and biology rather than to pathology. However, since the pathological changes in the gingival tissues and the periodontal membrane cannot be understood without a thorough knowledge of normal conditions, it was indispensable to enlarge upon the latter before entering into the question of the former.

The purpose of this book then may be characterized as follows: to illustrate by means of human specimens the actual tissue changes that correspond to certain well-defined, clinical conditions. For

example, a cavity is prepared and a filling is inserted. What tissue changes can be expected to follow this operation? The radiograph shows an area of bone destruction at the root end of a pulpless tooth. What tissues and cell forms are found in this area? A pulp is removed and the root canal is treated and filled. How will the periapical tissue react to this procedure? A bridge is cemented on abutments, thereby increasing the occlusal stress exerted upon these teeth. What will be the immediate and the later changes in the soft and hard tissues about the roots of the abutments? A tooth is moved orthodontically. What are the bone changes that make possible the movement of the tooth and its retention in the desired position? The author hopes, by introducing the clinical aspect of these problems instead of merely describing microscopic specimens, to facilitate the understanding of many clinical manifestations and practical observations.

The field of dental and oral pathology is so large that it is impossible to treat any particular subject in detail. Some problems have been well described and illustrated in earlier publications and textbooks. Others are relatively new and unknown to the profession; this latter group will be considered here more in detail.

The majority of the specimens described in this book has been taken from man. Animal tissues were used only to illustrate changes that were produced experimentally. All illustrations, except the diagrams, are photomicrographs, the author believing that, for the sake of objectivity, photographic reproductions are preferable to drawings. Unless otherwise indicated, the specimens from which the photomicrographs were taken belong to the collection of the Research Department of the Chicago College of Dental Surgery, Dental Department of Loyola University.

I wish to thank Dr. W. H. G. Logan for the helpful encouragement he has given this work, and to express my appreciation to Drs. R. W. Bunting, E. D. Coolidge, and E. B. Fink for the many valuable suggestions that I received from them and incorporated into this book. I also wish to express my gratitude to Miss Maurine Willman, who skilfully prepared all the necessary histological specimens and assisted in the preparation of the manuscript.

R. K.

CHICAGO, ILL.

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HISTOPATHOLOGY OF THE TEETH.

CHAPTER I.

ANOMALIES IN SHAPE AND NUMBER OF TEETH.

THE normal human deciduous dentition consists of twenty teeth; the permanent dentition, of thirty-two. These teeth are divided according to their shape and position in the arches into incisors, cuspids (canines), bicuspid (premolars), and molars. On the whole, the number and shape of the human teeth are remarkably constant; occasionally, however, abnormalities are observed that have some practical importance since they may require intervention on the part of the dentist or oral surgeon. These abnormalities can be loosely grouped into deviations from the normal shape and deviations from the normal number.

ANOMALIES IN THE SHAPE OF TEETH.

The measurements of anatomists and dentists have determined the averages for the length and width of the crown and root of the different tooth groups. Slight deviations from these averages are within the range of individual variation. Gross deviations are described as macrodontia, a condition in which the teeth are excessively large, and as microdontia, in which the teeth are abnormally small.

Macrodontia.—It is doubtful whether true, general macrodontia exists. Occasionally there are individuals with abnormally large teeth that mar the person's appearance as well as the balance of the dental arch. More often, however, macrodontia is only relative: although the teeth are actually not larger than the upper limits of physiological variation, they appear excessively large because the individual's face is narrow and the dental arches small. Such teeth are often crowded because of lack of space.

This condition usually has a hereditary background; the individual may have inherited a large tooth size from one parent and a narrow face and frail skeleton from the other.

Microdontia.—Microdontia may affect all teeth, or only single teeth, or a tooth group. In general microdontia, the teeth are small, the crowns short, and the normal contact points between the teeth are frequently missing. Sometimes this condition is the result of inheritance, namely, a combination of small tooth size with a large face and wide dental arches.

True microdontia of individual teeth is usually observed in those teeth that are also most likely to be congenitally missing, namely, the upper lateral incisors and the upper and lower third molars. In these teeth, microdontia is a transitional stage between normal development and absence of the whole tooth. Like the absence of certain teeth, microdontia seems to be inherited; there is a tendency for it to be transmitted from parents to children and to run in families.

Microdontia of the upper lateral incisors may result in teeth of fairly normal shape, but much narrower than normal. Or they may

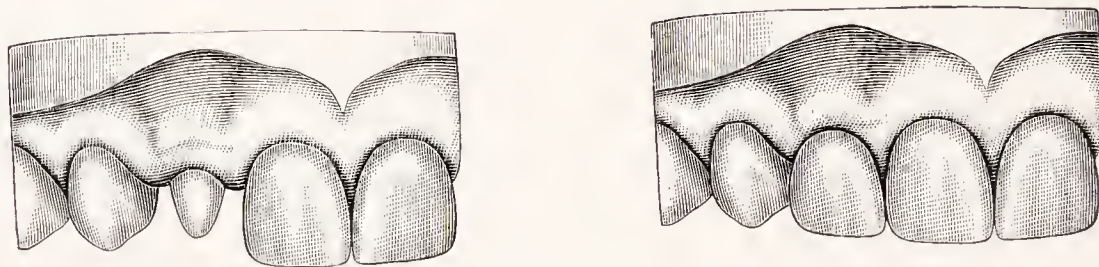


FIG. 1.—Conical lateral incisor covered by porcelain jacket crown. (Burchard and Inglis.)

be peg-shaped, being narrower at the incisal edge than in the cervical portion; then no contact points exist between these incisors and the adjoining teeth. Such teeth can often be successfully restored by placing porcelain jacket crowns upon the undersized crown (Fig. 1).

In the third molars, microdontia may vary from a mere reduction in the number of cusps to small, peg-shaped teeth that barely resemble a molar. Such teeth usually have a short, round, blunted root.

Fusion, Gemination, and Concrescence.—Fusion of teeth is a condition in which the crowns of two adjacent teeth, for instance, incisors, are united by a common covering of enamel (Fig. 2). The line of union is usually visible as a groove in the crown. The roots of the fused teeth may also be fused, or they may be separate. As a rule, there are two distinct pulp chambers, although occasionally one large, irregular pulp cavity may extend through the fused teeth. Another type of fusion occurs in the upper molar region where a second molar may be fused to a small supernumerary molar or to a dwarfed third molar (Fig. 3).

The etiology of fusion, like that of all congenital anomalies, is unknown.

The term gemination or twin formation is applied to the condition in which there is a double crown attached to a single root. For some unknown reason this condition is quite common in the deciduous dentition; a lower deciduous incisor, for instance, may have a

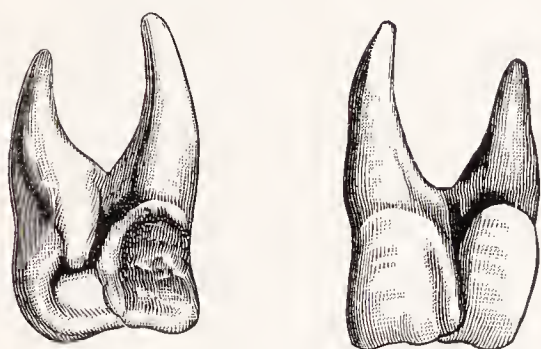


FIG. 2.—Fusion of the crowns and a portion of the roots of two permanent upper central incisors. (Burchard and Inglis.)



FIG. 3.—Fusion of a supernumerary tooth with an upper third molar. (Burchard and Inglis.)

crown twice as wide as normal, with a shallow groove running through the center of the incisal edge. In such instances, the crown is double, but the root remains single (Fig. 4). Sometimes the twin formation is not quite so distinct, and it is then impossible to distinguish between gemination and supernumerary cusps.

Concrescence is a coalescence of the cementum of the roots of two adjacent teeth. Most instances of concrescence are discovered during extraction when only one tooth is grasped with the forceps and two teeth are actually extracted. Sometimes one of the two teeth has remained unerupted. Both teeth have intact, independent crowns; the roots were originally separate and were united only secondarily as the cementum of the two roots fused (Fig. 5).



FIG. 4.—Gemination of upper central incisors. (Prinz.)

In sections through an area of concrescence it can be seen that the dentin of the two roots is separate, and only the covering of cementum is common to both, whereas in fusion the dentin of the two teeth is continuous.

Dens in Dente.—A peculiar dental anomaly that has received considerable attention recently is known as *dens in dente* (“one tooth within another”). The name indicates that one tooth is located within another. In reality such a condition has never been reported.

All the cases of dens in dente that have been studied histologically (Beust, Höpfel, Kitchin, Kronfeld) were merely deep invaginations of the lingual pit of an incisor into the rest of the crown. The result is a cavity inside of the tooth lined with enamel and opening on the



FIG. 5.—Concrescence of teeth. (Bunting.)



FIG. 6

FIG. 6.—Radiograph of a specimen of dens in dente. (Kitchin, Jour. Dent. Res.)



FIG. 7

FIG. 7.—Longitudinal ground section through specimen shown in Fig. 6. In the center of the crown there is a cavity, lined with enamel and opening at the tip of the tooth. (Kitchin, Jour. Dent. Res.)

lingual side of the crown (Figs. 6 and 7). The tooth thus affected is thick and deformed, and often its pulp is infected and gangrenous.

Anomalies in the Crown.—The crown of a tooth may lack a cusp that is present normally, or it may have additional or supernumerary

cusps. Closely related to the latter anomaly are the enamel drops or enamel pearls. They are small islands of enamel located on the root surface close to the cemento-enamel junction (Fig. 8). Enamel drops vary from microscopic size to 1 or 2 mm. in diameter; they may be single or multiple. Göllner, who studied their incidence in a large number of teeth, found them in 54 per cent of molars, 6 per cent of incisors, and in 15 per cent of all human permanent teeth. Frequently small enamel drops are covered by cementum and then can be seen only in histological sections.

Enamel drops owe their existence to an activity of the ameloblasts in a circumscribed area rootwise from the cemento-enamel



FIG. 8.—Enamel pearls (enamel drops). (Bunting.)

junction. There is no known reason for such an activity. They are of no clinical significance.

An anomaly occasionally encountered in bicuspid and molars is cementum in the occlusal fissures. This coronal cementum, as it is called, is laid down by the cementum-forming cells of the tooth follicle prior to the eruption of the crown. It is a rather common occurrence in embedded teeth but may also be found in teeth that have erupted normally (see Chapter X).

Anomalies in the Root.—The root, like the crown, may be unusually large or unusually small. Reduction in the size of the root may be due to various causes. Traumatic injuries during the developmental period may prematurely terminate its growth. The for-

mation of short, truncated roots may be the result of systemic disturbances, such as rickets or cretinism. Sometimes shortness of roots is hereditary. An unusual dental anomaly, hereditary opalescent dentin, in which short, thin roots are found, will be discussed later.

Accessory roots are very common. Additional roots have been found on every tooth from the incisors to the molars; sometimes there are as many as five or six thin roots on molars (Figs. 9 and 10).



FIG. 9

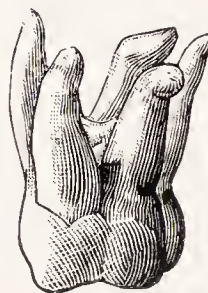


FIG. 10

FIG. 9.—Upper cuspid with two roots. (Burchard and Inglis.)

FIG. 10.—Upper third molar with five roots. (Burchard and Inglis.)

Hereditary Opalescent Dentin.—Hereditary opalescent dentin is a malformation that follows a strictly hereditary pattern. The teeth have short, squarish crowns with very prominent proximal convexities. Their color varies from gray to brown or bluish-brown. In

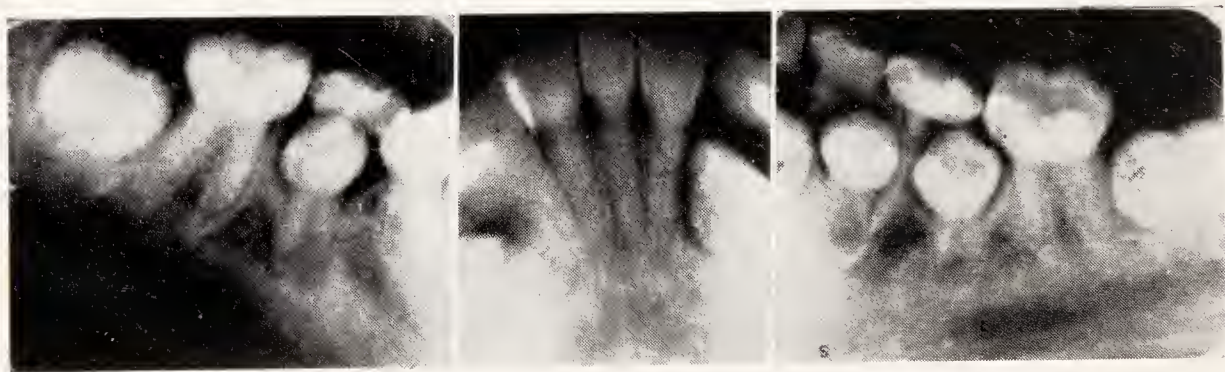


FIG. 11.—Radiographs of the mandibular teeth of a child, aged seven, with hereditary opalescent dentin. The teeth are grayish-brown and translucent. Note the absence of pulp chambers in the lower incisors, bicusps, and first permanent molars. The molar roots are short and pointed. Most of the members of this family were similarly affected. (Courtesy of O. J. McCormack.)

certain lights the teeth are transparent and show a faint iridescence or opalescence. They are usually soft, so that they soon wear down extensively.

Radiographs of such teeth reveal striking anomalies (Fig. 11). The pulp chamber is completely obliterated by calcified material. The roots are thin, short, and pointed, and the root canals, like the pulp chambers, are calcified. Histologically, the dentin shows an

irregular structure; the number of tubules is reduced, and the few tubules present are irregular and sometimes greatly enlarged. Often they are completely effaced by calcified material. The pulp cavity is filled with irregular dentin. The enamel appears normal (Fig. 12).

Hereditary opalescent dentin has been known as a pathological condition for the last fifty years. Recently a considerable number of cases has been reported, and probably many more observed. The hereditary nature of the anomaly is striking. In a number of families, hereditary opalescent dentin has been traced through several generations. Hodge and Skillen recently reported clinical and laboratory studies of this anomaly, including the histological investigation of the dental tissues; and Finn wrote a complete review of the literature on the subject.

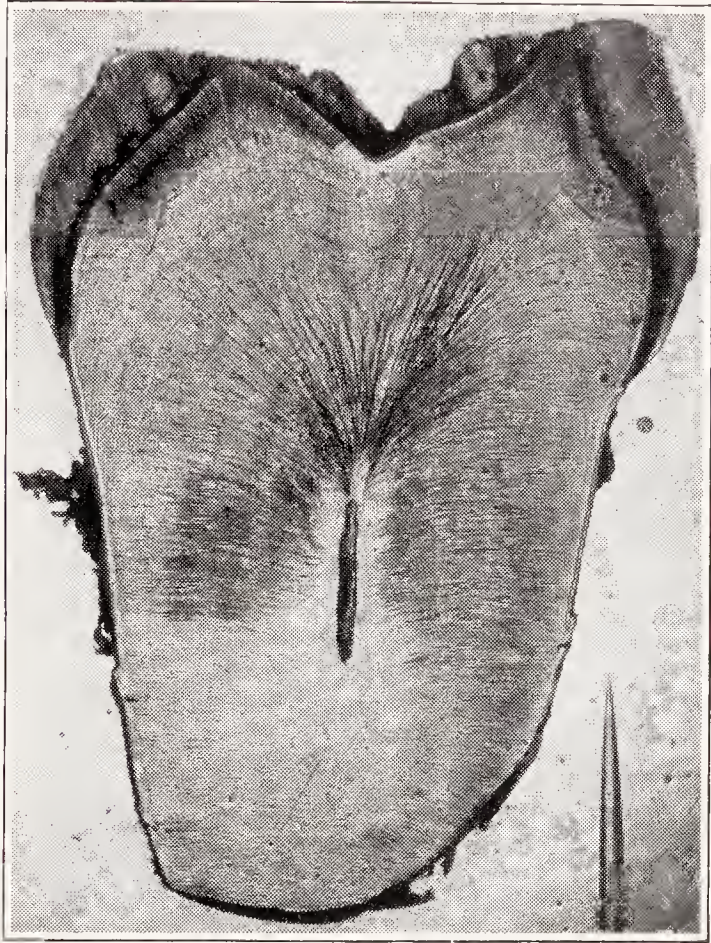


FIG. 12.—Ground section through a molar of a patient with hereditary opalescent dentin. The dentinal tubules are irregular; the pulp cavity is reduced to a narrow slit. (Skillen, Jour. Am. Dent. Assn.)

The etiology of the condition is obscure. Careful physical and laboratory examinations of the patients have failed to reveal any significant deviations from the norm. All that is known is that hereditary opalescent dentin is a dominant hereditary factor, affecting approximately one-half of the offspring, without preference for either sex. The prognosis for such teeth is rather bad; because of the poor structure and the rapidity with which they wear down, most of the affected teeth are destroyed by caries or extracted early in life.

ANOMALIES IN THE NUMBER OF TEETH.

Any teeth in excess of the thirty-two of the permanent dentition are called supernumerary teeth. Contrariwise, teeth or tooth groups that fail to develop are said to be congenitally missing. In its extreme form the latter condition is called anodontia: all, or nearly all, teeth of the permanent set are lacking.

Supernumerary Teeth.—Supernumerary teeth can be divided into two major classes: teeth resembling the normal tooth form, and small peg-shaped teeth that bear no resemblance to any normal tooth form. Supernumerary teeth may occur anywhere in the dental arch; however, there are certain places where the majority of them are found. One of these areas is the region of the upper central incisors. One or two round, small, peg-shaped supernumerary teeth may be found near the midline, located either in the bone above the roots of the central incisors, or erupted behind or between the central incisor teeth. This form of supernumerary teeth was called *mesiodens* by Bolk.

Supernumerary central and lateral incisors have occasionally been observed. Supernumerary cuspids occur, but are extremely rare. Supernumerary bicuspid, on the other hand, are quite common. Sometimes they occur bilaterally, so that an individual may have three

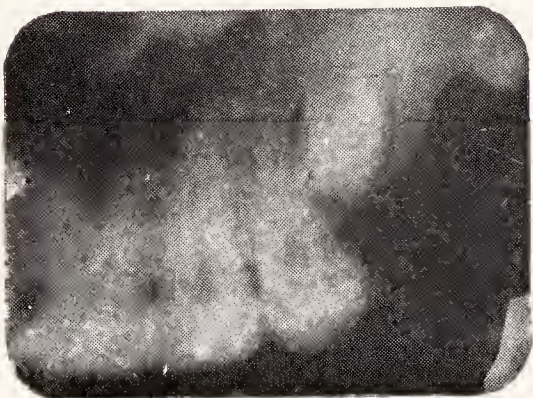


FIG. 13

FIG. 13.—Radiograph of embedded upper fourth molar. (Munoz, *Dent. Cosmos*.)



FIG. 14

FIG. 14.—Two large supernumerary upper incisors erupted on the lingual side of the normal incisors. (Stafne, *Dent. Cosmos*.)

lower bicuspid on both sides of the mandible. Supernumerary molars are occasionally found at the end of the dental arch and are known as fourth molars (Fig. 13). Usually they are smaller than normal molars, and because of lack of space in the dental arch they frequently remain embedded.

In the clinical and radiographic study of supernumerary teeth it is advisable to distinguish between erupted and embedded supernumerary teeth. The erupted ones often cause crowding and malocclusion, and therefore their extraction is indicated. Frequently after the removal of supernumerary teeth, orthodontic treatment is necessary to correct the rotated or otherwise abnormal position of the remaining teeth (Fig. 14).

Embedded supernumerary teeth can be diagnosed only by means of the radiograph. Many of them cause no symptoms or disturbance and are discovered by a routine radiographic examination; they should be left alone. The surgical removal of an embedded supernumerary tooth is indicated only if it interferes with the normal alignment of the adjacent erupted teeth. This may occur, for instance, if an embedded mesiodens lies between the roots of the upper central incisors and causes an abnormal space (diastema) between these teeth. Obviously, attempts to reduce this space orthodontically are bound to fail unless the mesiodens that keeps the roots apart is first removed.

There may be resorption of enamel and dentin in embedded supernumerary teeth, which may lead to almost complete destruction of the tooth and its replacement by bone (Stafne).

Congenitally Missing Teeth. Anodontia.—If one or several teeth are missing and there is no history of their loss or extraction, two possibilities have to be considered: either the teeth were formed but failed to erupt, or they failed to form. The decision as to which one of these conditions exists can be made only by means of the radiograph.

Multiple Unerupted Teeth.—If there are no mechanical obstacles, failure of several teeth to erupt often indicates some systemic disturbance. The disturbance may be hypothyroidism (cretinism), or it may be cleidofacial (cleidocranial) dysostosis. In the latter, a peculiar systemic anomaly, the clavicles are missing or are present only as short stubs; as a result, the individual is able to bring the shoulders together in front of the chest. Most of the permanent teeth remain embedded in the jaws; only a few erupt. Frequently the number of teeth is greater than normal because of several supernumerary teeth. In the case of a family studied by Heupel, cleidofacial dysostosis was present in the father and in three girls out of eight children; the other two girls and three boys were normal in every respect.

Little can be done to help patients with multiple unerupted teeth. In endocrine disturbances, treatment with the hormone in which the patient is deficient will sometimes stimulate eruption of the embedded teeth. In cleidofacial dysostosis the only treatment is the construction of suitable dentures. In the case mentioned above the father of the family, a man in his fifties, had been wearing full dentures for the greater part of his life, although his jaws contained at least twenty-four unerupted teeth.

Congenitally Missing Teeth.—Congenitally missing teeth are the result of a failure of the tooth germ to form or to develop. This

may happen to any tooth germ; however, some teeth are far more frequently affected than others. Congenital absence of the permanent cuspids or of the first permanent molars is very rare; absence of the upper lateral incisors, bicuspid, or third molars, however, is not uncommon. Particularly the upper lateral incisor is frequently missing, often on both sides; as a result, the cuspids are in contact with the central incisors (Fig. 15).

Like microdontia, congenital absence of teeth (anodontia) follows a hereditary pattern. Trauner and Preissecker studied two families in which the absence of numerous permanent teeth was found in three generations; it appeared as a dominant hereditary characteristic. The upper lateral incisors and the third molars were the teeth most frequently missing. Dahlberg reported the absence of anterior teeth in 18 members of a family, distributed over four generations.



FIG. 15.—Plaster casts of a young adult with congenitally missing upper lateral incisors. In spite of the absence of these teeth, alignment and occlusion are quite satisfactory.

Occasionally, absence of teeth is associated with other congenital malformations. Among 50 patients with congenital absence of teeth Willner found 16 who had other malformations of ectodermal structures, especially of the hair, fingernails, and sebaceous and sweat glands. Mathis observed a brother and sister both of whom had partial anodontia and congenital malformation of the iris.

Complete anodontia or absence of all teeth is very rare; more often a few teeth are present, usually lower incisors or first molars. Thoma, in a review of this subject, emphasized that even in complete anodontia the mandible is of normal length and its general development is not disturbed, except for the absence of the alveolar process.

A good example of almost complete anodontia is the patient described by Porter and Edwards. A girl, aged thirteen years, who otherwise appeared physically and mentally well developed, had only eleven deciduous teeth and three permanent teeth, both upper

first permanent molars and the upper left second permanent molar (Fig. 16). In this case there was complete anodontia in the permanent dentition in the mandible, and partial anodontia in the maxilla.

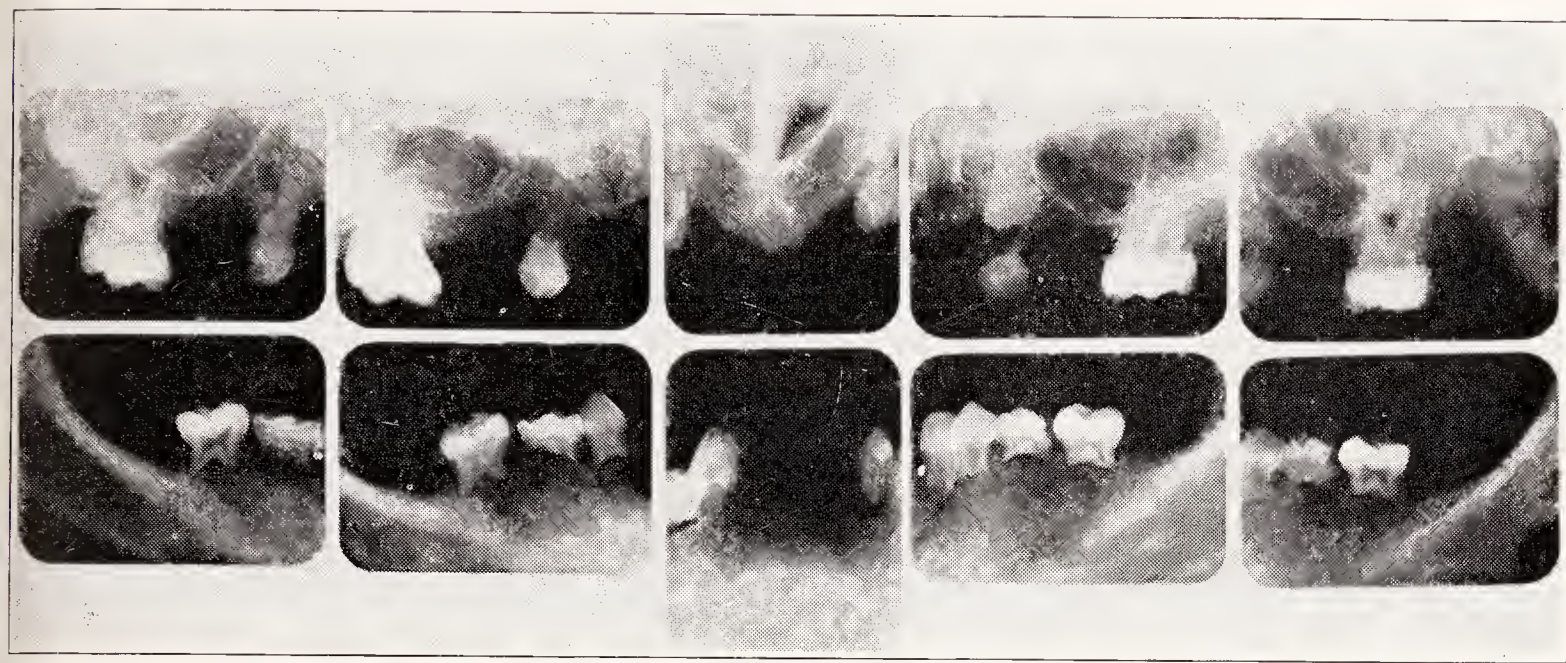


FIG. 16.—Radiographs of a girl, aged thirteen years, with almost complete anodontia. The only teeth present are several deciduous teeth and three upper permanent molars. Note the extensive resorption of the deciduous teeth in spite of the absence of permanent teeth. (Porter and Edwards, Jour. Am. Dent. Assn.)

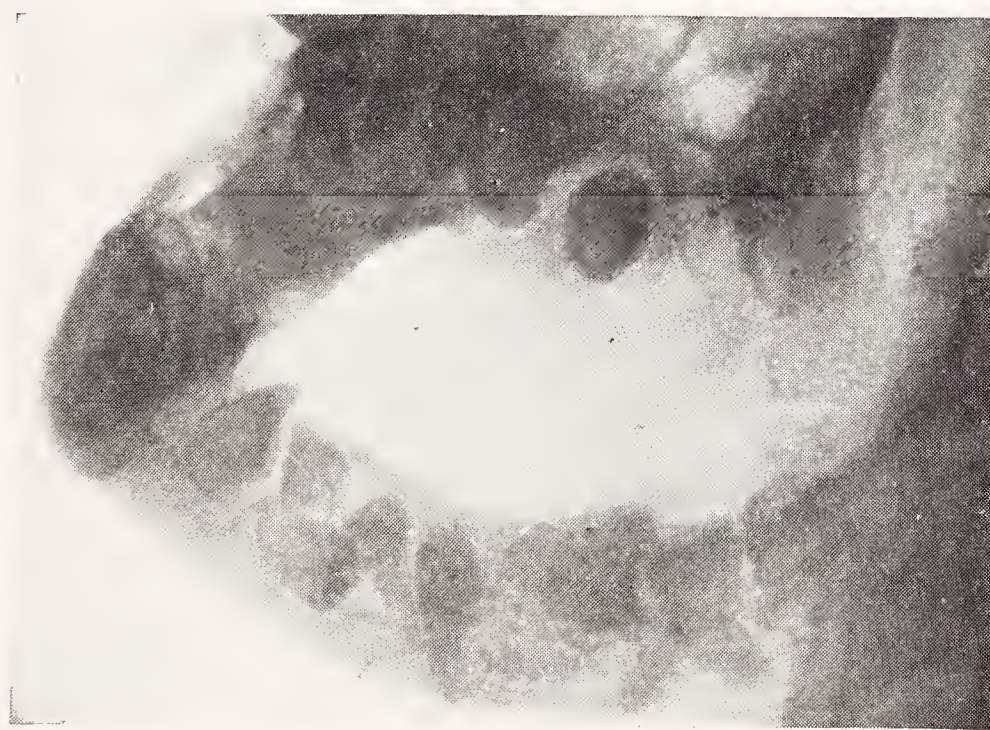


FIG. 17.—Radiograph showing multiple unerupted teeth in the edentulous mandible of a woman, aged twenty-eight years. (Ivy, Dent. Cosmos.)

Anodontia must not be confused with multiple retention of teeth in the jaw. Ivy described a patient's mouth which upon superficial examination resembled the case illustrated in Figure 16; yet the radiograph revealed many unerupted teeth lying in the jaw bone (Fig. 17).

ANOMALIES IN ERUPTION.

The data for the normal eruption of the deciduous and permanent teeth are given in the table on page 38. There are considerable individual variations in the time of eruption, depending upon geographical location, social conditions, race, and sex. However, there are also variations extreme enough to be called pathological; they can be classified into premature eruption and delayed eruption.

Premature Eruption.—On rare occasions one or two deciduous teeth have been found erupted in new-born infants. Such teeth usually have to be removed since they interfere with nursing. They have very short roots and are loosely attached to the jaw ridge.

Premature eruption of the permanent teeth occurs, especially in the bicuspid region, because of the early extraction of the deciduous teeth. Bicuspid may erupt at the age of eight years or even earlier (Fig. 18). The roots of such prematurely erupted teeth are still

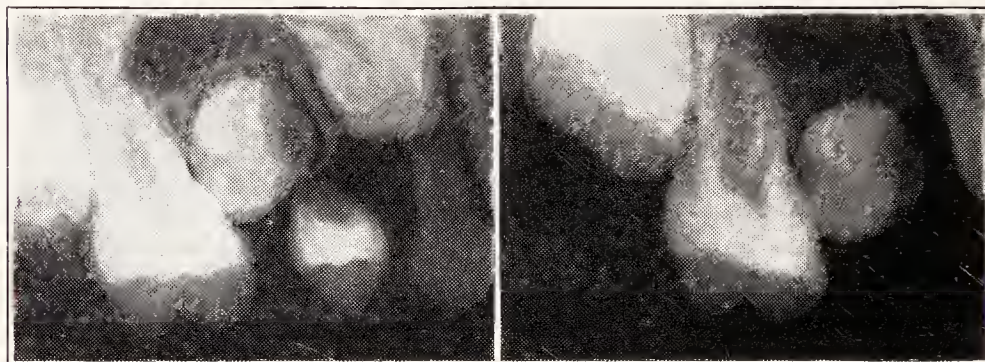


FIG. 18.—Left: Premature eruption of bicuspid in a child, aged eight years, caused by loss of deciduous molars. Right: Same area four months later. The first bicuspid was spontaneously exfoliated because of lack of root. The second bicuspid is malposed. (McCall, Jour. Am. Dent. Assn.)

very short; often only the cervical one-third of the root has been formed. Root formation may then continue normally, or the newly erupted tooth may become progressively looser and finally be lost.

Delayed Eruption.—The delay in eruption may be general, affecting the entire dentition, or it may affect only one or two teeth. If the eruption of the deciduous incisors is delayed much beyond the age of one year, it is very likely that the infant has rickets or that some developmental disturbance is present. The same is true of the eruption of the permanent teeth: endocrine disturbances, such as cretinism, may delay their eruption for several years.

As far as individual permanent teeth are concerned, eruption may be greatly delayed by delayed exfoliation of the deciduous predecessor. A bony scar on the occlusal side of a permanent tooth germ may also retard eruption; such a scar may be the result of an injury to or an infection of the deciduous tooth.

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CHAPTER II.

PHYSIOLOGY AND PATHOLOGY OF TOOTH CALCIFICATION.

IN this chapter the most important problems of calcium metabolism and calcification will be given in condensed form. Special attention will be paid to the influence of nutrition upon tooth structure, the biological difference between teeth and bone, the possibility of calcium withdrawal, and the incidence of caries in pregnancy.

CALCIFICATION OF TEETH.

Normal Calcification of Teeth and Bones.—Calcification is the transformation of soluble calcium salts into insoluble ones (mainly calcium phosphates) and their deposition into the tissues of the body. The original source of calcium is the various foodstuffs that constitute the daily diet. This calcium of the food is absorbed through the small intestine and, in a soluble form, appears in the blood serum. Under physiological conditions, the amount of calcium in the human blood serum is from 9 to 11 mg. per 100 cc. This amount is fairly constant; it is regulated by the hormone of the parathyroid gland.

In areas of physiological calcification, such as in forming bone or forming dental hard tissues, a matrix or ground substance is present that has a special affinity to calcium salts. The soluble calcium salts of the blood serum are precipitated into this ground substance and are transformed into insoluble calcium phosphate. As far as is known, calcium and phosphorus are combined in the hard tissues to form an empirical formula: $3\text{Ca}_3(\text{PO}_4)_2 \cdot \text{Ca}(\text{OH})_2$. This closely resembles a substance known in mineralogy as hydroxylapatite.

In addition to the parathyroid hormone, vitamin D plays an important part in the normal process of calcification. A sufficient supply of this vitamin is necessary to insure proper calcification. In vitamin D deficiency the matrix of bone and teeth is formed, but only a small amount of calcium salts is deposited in it, and the bone or tooth remains poorly calcified. One of the symptoms of rickets is insufficient calcification of the bones and teeth.

In pathological calcifications, the mechanism is the same, except that the predisposing factors vary. Regardless of the tissue that

is being calcified, whether it is a calcifying tubercle, a bone tumor, or a pulp stone, at first there is always some soft structure or matrix into which the lime salts are deposited.

Fundamental Differences Between Bones and Teeth in Regard to Calcification.—The bones act as a storehouse for calcium, which is deposited in them when the supply is ample and is withdrawn when there is need for it. The bones of the adult skeleton are constantly being resorbed and rebuilt. In youth, during growth, more calcium is deposited in the skeleton than is withdrawn. During adult life, calcium deposition and calcium withdrawal are about equal. In old age, more calcium is withdrawn than deposited, so that the bones become porous and brittle.

Calcium is withdrawn from the bones under certain pathological conditions, such as osteomalacia or excessive activity of the parathyroid glands. In osteomalacia the formerly normal bones are rapidly decalcified and become soft. Large quantities of calcium are eliminated in the urine and feces. As a result, extensive deformities of the skeleton develop; the bones bend under the influence of muscle pull and body weight, and the patient becomes deformed and crippled. Similar conditions develop when the parathyroid glands are overactive. Calcium is drained from the skeleton; the bones are resorbed and replaced by fibrous tissue, and multiple fractures of the weakened bones occur.

For the most part the dental hard tissues are calcified during the developmental period, prior to eruption; only in the dentin and cementum does calcification of newly formed matrix continue throughout life. *During their development the human teeth are very sensitive to variations in the calcium supply, but this is not true of adult, erupted teeth.* Calcium cannot be withdrawn from the enamel by tissue metabolism; bone may become decalcified by osteoclastic resorption or by direct dissolution of the calcium salts, but no such process has ever been demonstrated in the human teeth.

Thus, the differences between bone and teeth can be summarized as follows: in bone there is a continuous process of calcification and calcium withdrawal; in the teeth calcification only occurs, there being no calcium withdrawal comparable to that from the bones.

Negative Evidence Concerning Calcium Withdrawal from the Teeth.—So far it has not been convincingly demonstrated that calcium or any other substance can be drained from human teeth. On the contrary, there is evidence that the calcium in the teeth is very stable, even when large amounts of it are being withdrawn from the skeleton. A few of these observations will be reviewed here.

Gies demonstrated in dogs the effect of intraperitoneal injections of trypan blue at different stages of dental development. If trypan blue was injected before the permanent teeth had begun to form, all of the enamel of the latter was stained blue. If trypan blue was injected soon after the permanent teeth had begun to form, the teeth were white except for a blue zone in the cervical portion. If the injections were made after the enamel of the permanent teeth was fully formed, the enamel remained white, whereas the pulp and adjacent dentin stained blue. Some of the dogs with blue enamel were kept under observation for years. The blue color of the teeth did not change, although the dye was gradually eliminated from all other tissues. Sections through such teeth showed the dye in both dentin and enamel. From these findings Gies suggested that substances circulating in the blood during the period of enamel development are incorporated into the enamel and retained there indefinitely.

Fish kept a pregnant dog on a diet that was very poor in calcium. At the beginning of the experiment a tooth was extracted, and the amount of calcium in it determined. At the end of the experiment the bones were soft and decalcified to such an extent that they were hardly visible in the radiograph and could be cut with a knife; the teeth, however, showed the same density radiographically as before, and a chemical analysis showed the calcium content unaltered.

By a chemical analysis of the dentin of many human teeth from individuals of different ages, Fish found that there is a gradual, slight increase in the calcification of the dentin with advancing age, but never a calcium loss. He came to the following conclusions: "It has not been found possible to modify the calcium content of the dentin either by parathyroidectomy, calcium-deficient diet, pregnancy, or feeding on therapeutic or excessive doses of vitamin D; nor did two dogs with extreme natural softening of the bones show any loss of calcium from the dentin. It appears, therefore, that the calcium content of the dentin is extremely stable, that slight physiological additions may be made to it, but that there is no evidence as yet of any definite loss of calcium from the dentin under any circumstances."

Albright, Aub, and Bauer reported the clinical and laboratory findings in 17 patients with hyperparathyroidism and decalcification of the bones. They state: "The teeth do not take part in the generalized decalcification. They may fall out because of disease of the jaws but they themselves remain well calcified. This is brought out strikingly by roentgenograms in which the well calcified

teeth stand out sharply against the poorly calcified jaws. This failure of the teeth to become decalcified is strong evidence against their being a reserve supply of calcium."

Recently Hatton studied the teeth in gastrectomized dogs. In dogs, surgical removal of the stomach produces a porous condition of the bones and loss of weight of the skeleton. The teeth, however, do not take part in this calcium withdrawal but remain as highly calcified as they were before the operation.

These few examples may suffice to show that there is no justification for assuming that the teeth are a reserve supply of calcium or that calcium can be withdrawn from them. Until some new evidence is brought forth, the conclusion must be that the calcium content of the enamel does not change once the tooth is erupted, and that the calcium content of the dentin also remains stable, or increases slightly during life, but never decreases. *Unlike the bones, the teeth are not subject to calcium withdrawal.*

Influence of Diet Upon the Structure and Composition of Adult Teeth.—From the foregoing it is evident that the only tooth whose structure and calcification can be influenced by dietary means is the *growing* tooth. While the tooth structures, especially the enamel, are being formed, a well-balanced diet rich in minerals and vitamin D is necessary to insure proper calcium supply to the body and thus to the developing teeth. After the tooth has erupted, no internal changes can be expected from dietary measures. A nutritionally sound diet is, of course, desirable and necessary throughout life to keep all tissues and structures, including the bones, supplied with their needs; but so far as the composition of the adult teeth is concerned, the diet is of no significance.

Much confusion has been created by a failure to distinguish clearly between *growing* teeth and *adult* teeth. In certain animals with continuously growing teeth, for instance in the rat, the teeth can be influenced by dietary means *because there is always a portion of the tooth that is growing*. In the adult teeth of man, which are no longer growing in the usual sense of the word, the situation is basically different.

Recently, a number of calcium preparations have been put on the market, and dentists and patients have been led to believe that the use of such preparations can improve the structure of the teeth and decrease dental caries and other dental disorders. Such hopes are unfounded. Very little of this artificial calcium supply is actually assimilated, and whatever is assimilated cannot be of any benefit to adult teeth, especially not to the enamel. All of this has been

brought out clearly in a report of the Council on Dental Therapeutics of the American Dental Association. Apparently this report has not received the attention that it should have, and some dentists still recommend calcium preparations to their patients in the hope of arresting caries.

The report of the Council on Therapeutics reviews the subject of the use of calcium and phosphorus compounds in dentistry. Some of the conclusions are:

“There is no carefully controlled evidence that the addition of calcium and phosphorus compounds, whether inorganic or organic, promote retention of these elements and hence freedom from dental diseases, except in known cases of deficiency. Where this occurs, milk serves as an excellent source of calcium and phosphorus in a readily assimilable form.”

The opinion that an addition of calcium and phosphorus to the diet of a pregnant woman will influence or improve the teeth of the child does not stand up under a critical analysis. Hess and his collaborators came to the following conclusion concerning prenatal tooth calcification and prenatal calcium medication:

“It was found that at birth only a small quantity of calcium and phosphorus has been deposited even in the deciduous teeth. At this time, the total calcium phosphate laid down in the teeth of both jaws amounts to only about one-half a gram, which is 15 to 20 per cent of the content of the fully developed teeth. It is quite unnecessary and of little value to feed large amounts of calcium and phosphorus to mothers during pregnancy in order to bring about deposition of calcium in the teeth of the fetus.”

RELATIONSHIP BETWEEN CARIES AND PREGNANCY.

There is an old adage, “A tooth for every child,” expressing the belief that pregnancy has a detrimental effect upon the teeth. The usual explanation of this supposed correlation is that the teeth are decalcified to furnish calcium for the fetus.

If the problem of pregnancy and caries is analyzed, it resolves itself into two questions: Is there actually an increase in the amount of caries during pregnancy? and, if there is such an increase, how can it be explained?

The answer to the first question can be given only by statistical data. It is necessary to study caries incidence in a great number of pregnant women and then to compare the results with the caries incidence in an equal number of non-pregnant women of the same

ages, the same race, and the same social level. Only the results obtained in this way are reliable and can rightly be used for generalizations; individual observations may easily be misleading.

Ziskin made a statistical study of dental caries in more than 800 pregnant and non-pregnant women. He found a definite increase in the number of decayed and missing teeth with advancing age, but no correlation between the condition of the teeth and the number of pregnancies. The number of pregnancies had no influence upon the frequency with which the teeth decayed.

From the examination of the teeth of a large number of pregnant and non-pregnant women of identical age groups, Starobinsky came to the conclusion that the number of carious teeth is not greater in pregnant than in non-pregnant women, and that the gradual increase in the number of carious teeth in women with repeated pregnancies is not caused by the pregnancies, but by the increase in age.

Mull, Bill, and Kinney reported the dental findings in 358 women who were examined during pregnancy and after delivery. The incidence of new cavities during this period did not exceed the average caries incidence for all women of identical age range during the same length of time. Some of their conclusions are:

"There is no appreciable change in the teeth of women during pregnancy or the first few weeks of lactation other than that which would probably occur in a similar group of non-pregnant women during the same period of time. Only 15 per cent of the cases studied showed change.

"A general laxity in the oral hygiene was observed, especially following delivery. This condition would probably not be true in other social groups, and may or may not have any bearing on the condition of the teeth.

"There is no rise in the average number of missing and carious teeth with the number of pregnancies experienced. There is a distinct increase, however, with age. Examination of the teeth of unmarried women showed them to be no better than those of women who had borne one or more children.

"It is evident that the condition of pregnancy as observed in this area cannot be considered a primary cause of tooth destruction."

The second part of the problem is how a slight increase in caries incidence in pregnancy could be explained, if such an increase were actually demonstrated. The assumption of a metabolic calcium withdrawal from the teeth has been shown to be unfounded. But there are other factors that influence the oral conditions during pregnancy. One is the gingivitis that occurs in a large percentage

of pregnant women and that increases retention of food débris. Then there is the general laxity in oral care that so often accompanies pregnancy. Mull, Bill, and Kinney recorded the observation that, especially following delivery when the care of the child makes an increased demand upon the mother's time, less and less attention is paid to the care of the mouth. This might increase the incidence of decay in a caries-susceptible mouth.

From a clinical point of view the problem of pregnancy and the teeth can be summed up as follows: No pregnant woman who takes proper care of herself, who practices ordinary oral hygiene and, if necessary, counteracts the bad effects of gingivitis by proper treatment and regular attention to her mouth, needs to lose a single tooth, even with repeated pregnancies. Contrary to public opinion, there is no reason why a pregnant woman should not have restorative dental work done.

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CHAPTER III.

ENAMEL HYPOPLASIA. HUTCHINSON'S TEETH. MOTTLED ENAMEL.

CLINICAL MANIFESTATIONS AND ETIOLOGY OF ENAMEL HYPOPLASIA.

ENAMEL hypoplasia is caused by a defective formation of the enamel of the deciduous and permanent teeth. It is present when the tooth erupts and persists unchanged throughout life. In this respect it differs basically from such defects as caries, abrasion, or erosion which develop in a formerly intact tooth after it has erupted.

The clinical appearance of enamel hypoplasia varies widely. In mild cases, the enamel is intact except for several shallow depres-



FIG. 19.—Enamel hypoplasia. (Bunting.)

sions or grooves on otherwise smooth enamel surfaces; in more extensive cases, the grooves or pits are arranged in horizontal rows around the crown, extending into the enamel as far as the dentino-enamel junction (Fig. 19). In severe cases, the enamel on the incisal edge of the incisors and cuspids is lacking, and the dentin on the occlusal surface of the first permanent molar is exposed. The small amount of enamel present in such cases is distributed irregularly on the labial and lingual surfaces of the teeth.

The term “enamel hypoplasia” was first used by Zsigmondy. It is preferable to the old term “atrophy” because the condition is characterized by an underdevelopment of the enamel, while the word

CHRONOLOGY OF THE HUMAN DENTITION.
Logan and Kronfeld (Slightly Modified by McCall and Schour).

Tooth.		First evidence of calcification.	Enamel completed.	Eruption.	Root completed.
<i>Deciduous dentition</i>	Central incisor	4 mos. in utero	4 mos.	6-8 mos.	1½-2 yrs.
	Lateral incisor	4½ mos. in utero	5 mos.	8-10 mos.	1½-2 yrs.
	Cuspid	5 mos. in utero	9 mos.	16-20 mos.	2½-3 yrs.
	First molar	5 mos. in utero	6 mos.	12-16 mos.	2-2½ yrs.
	Second molar	6 mos. in utero	10-12 mos.	20-30 mos.	3 yrs.
<i>Permanent dentition</i>	<i>Upper jaw</i>	Central incisor	3-4 mos.	7-8 yrs.	10 yrs.
		Lateral incisor	1 yr.	8-9 yrs.	11 yrs.
		Cuspid	4-5 mos.	11-12 yrs.	13-15 yrs.
		First bicuspid	1½-1¾ yrs.	10-11 yrs.	12-13 yrs.
		Second bicuspid	2-2¼ yrs.	10-12 yrs.	12-14 yrs.
		First molar	at birth	6-7 yrs.	9-10 yrs.
		Second molar	2½-3 yrs.	12-13 yrs.	14-16 yrs.
	<i>Lower jaw</i>	Third molar	7-9 yrs.	17-21 yrs.	18-25 yrs.
		Central incisor	3-4 mos.	6-7 yrs.	9 yrs.
		Lateral incisor	3-4 mos.	7-8 yrs.	10 yrs.
		Cuspid	4-5 mos.	9-10 yrs.	12-14 yrs.
		First bicuspid	1¾-2 yrs.	10-12 yrs.	12-13 yrs.
		Second bicuspid	2¼-2½ yrs.	11-12 yrs.	13-14 yrs.
		First molar	at birth	6-7 yrs.	9-10 yrs.
		Second molar	2½-3 yrs.	11-13 yrs.	14-15 yrs.
		Third molar	8-10 yrs.	17-21 yrs.	18-25 yrs.

atrophy indicates a wasting or reduction in size of a fully-developed tissue or organ.

Enamel hypoplasia is the result of a pre-eruptive disturbance of the enamel formation. The enamel prisms in the involved areas remain permanently in the state of development they had reached when the disturbance occurred. After the interference subsides, normal enamel may again be formed. If the disturbance is repeated, a zone of missing or underdeveloped enamel will again result; hence, an alternating distribution of the hypoplastic areas on the surface of the crown is observed. From the chronology of the enamel formation of the human deciduous and permanent teeth, it is possible to estimate the age of the child at the time when the disturbance occurred.

Enamel Hypoplasia of the Deciduous Teeth.—The calcification of the deciduous teeth begins during the fifth and sixth months of intrauterine life. The enamel formation is well under way at the time of birth; the enamel of the deciduous incisors and first molars is completed between four and six months after birth, that of the deciduous cuspids and second molars between nine months and one year. Consequently, the enamel of all deciduous teeth can be divided into an inner, prenatal enamel and an outer, postnatal enamel. The dividing plane between these two layers is visible in most deciduous teeth; it has been described by Schour as the neonatal ring.

Because of the neonatal ring it has been possible to determine accurately the chronology of the deciduous dentition and also of enamel hypoplasia in deciduous teeth. Thus, it was found that hypoplasia of the deciduous teeth usually affects the postnatally formed enamel. Up to birth enamel formation is, as a rule, undisturbed and regular. The mother keeps the fetus amply supplied with all substances necessary for the proper formation of the tissues, and, as a result, the prenataally formed portion of the deciduous teeth is almost never abnormal.

After birth the situation is different. During the first few months of life many infants suffer from diseases and disturbances, and consequently the enamel formed during this period may be poorly calcified or hypoplastic. Then the neonatal ring is the division between well-formed prenatal and hypoplastic postnatal enamel.

Stein has pointed out that the deciduous teeth of prematurely born infants are frequently hypoplastic. The health of such infants is usually poorer than that of those born at term; they develop more slowly and are more subject to illnesses of all kinds. This

accounts for the higher incidence of disturbed enamel formation in their deciduous teeth. Schour and the author have described the dentition of an infant of seven months with a traumatic birth injury in whom enamel formation in most deciduous teeth had terminated at birth. The term "neonatal dental hypoplasia" was applied to this condition. In Figure 20 a ground section through a lower first

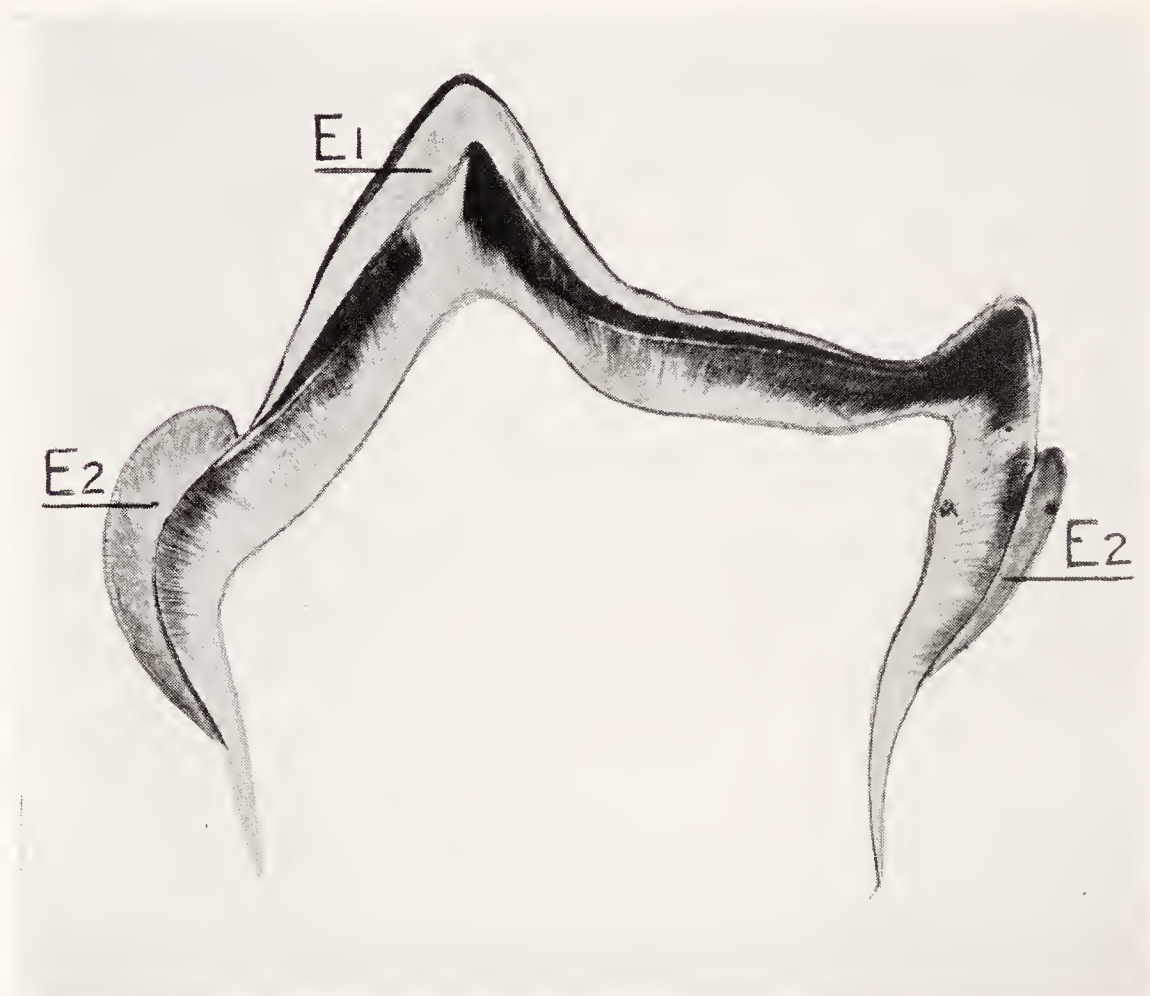


FIG. 20.—Ground section through the lower first deciduous molar of a child with birth injury and neonatal dental hypoplasia. In the occlusal portion of the crown enamel formation has terminated at birth; here only thin prenatal enamel, E_1 , is present. In the cervical portion of the crown, postnatal enamel, E_2 , has formed a circular ledge of normal thickness. (Kronfeld and Schour, Arch. Pathology and Jour. Am. Dent. Assn.)

deciduous molar of this infant is shown. The occlusal portion of the crown is covered with prenatal enamel only; here enamel formation terminated at birth. Postnatal enamel was laid down in the form of a circular ledge in the gingival one-half of the crown. In the dentin the neonatal ring is clearly visible.

Enamel Hypoplasia of the Permanent Teeth.—The calcification of the permanent teeth begins at birth in the first permanent molars. It is entirely postnatal, and hypoplasia of the permanent teeth is therefore always the result of a postnatal disturbance. The permanent central incisors begin to calcify at three to four months after birth, the lower lateral incisors at about the same time. Next

are the permanent cuspids whose calcification begins at four to five months. The permanent upper lateral incisors calcify at about ten months to one year. Calcification of the bicuspids begins between one and three-quarter years and two and one-half years, that of the second permanent molars from two and one-half to three years, and that of the third molars from seven to eight years.

Using the above data and the table on page 38 as a guide, it is possible to predict the result that a disturbance of the enamel formation at any given period of life may produce; likewise, from a hypoplastic area on an erupted tooth one can determine approximately when the damage to the enamel-forming tissues occurred.

If the interference with enamel formation takes place in the first year of life, the central incisors, the lower lateral incisors, and the

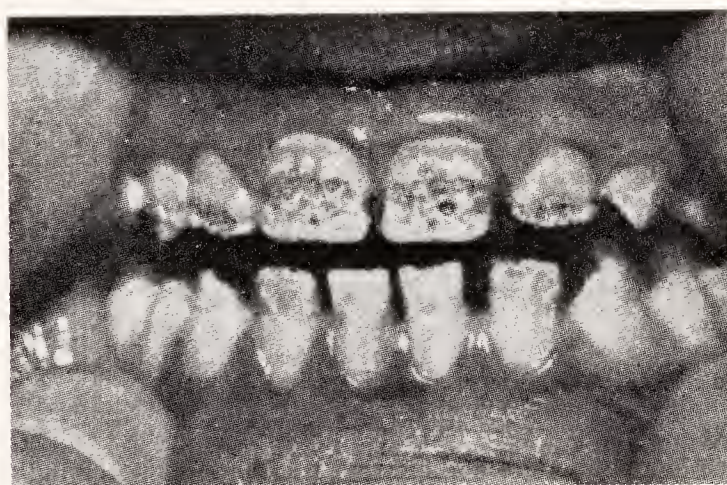


FIG. 21.—Photograph of the teeth of a child with severe rickets. (Eliot, *et al.*, *Am. Jour. Dis. Child.*)

first permanent molars will show evidence of enamel hypoplasia in the incisal or occlusal portion of the crowns. The cuspids are usually involved to a lesser extent than the incisors (Fig. 16). If the disturbance occurs in the second year, the upper lateral incisors will also be affected (Fig. 21). Although the cuspids erupt at the age of eleven or twelve years, they begin to calcify earlier than the upper lateral incisors, which erupt at the age of seven or eight years; microscopic findings have established beyond doubt the fact that the cuspids begin to calcify about six months before the upper lateral incisors. This is clearly borne out in those cases of hypoplasia in which the central incisors and the cuspids are defective while the upper lateral incisors are intact. The bicuspids and second permanent molars are affected only if the disturbance occurred or persisted during the third and fourth years of life or still later. Then the hypoplasia of the incisors and cuspids is usually very extensive, involving most of the crown. The third molar is seldom involved.

RICKETS, TETANY, AND ENAMEL HYPOPLASIA.

For a long time the etiology of enamel hypoplasia was obscure. Toward the turn of the century, rickets was generally held responsible for it. In 1909, Fleischmann came to the conclusion that rickets could not be considered the only etiological factor in enamel hypoplasia. He argued that rickets is by far more common than enamel hypoplasia and is, furthermore, a continuous, chronic disease, whereas the distribution of enamel hypoplasia points toward a disease with an intermittent course. Fleischmann believed tetany to be the cause, and he reported an analysis of ten cases of hypoplasia all of which gave a history of tetany (convulsions) at the time when the affected teeth were forming.

The investigations of Erdheim and Toyofuku cast new light upon the difficult problem of disturbed enamel calcification. Erdheim reported that the removal of the parathyroid glands in rats caused severe disturbances in the enamel formation of the incisors. The enamel epithelium proliferated, became detached from the enamel surface, and formed irregular, poorly calcified enamel masses. The similarity of the changes in parathyroidectomized rats to the findings in human jaw specimens suggested that human enamel hypoplasia is the result of a disturbance of the parathyroid glands at the time of enamel formation. As mentioned before, Fleischmann corroborated this idea with histories of patients in which there was a coincidence of tetany and hypoplasia of the enamel that was being formed at the time of the attack of tetany.

A few data will be given here concerning the pathology and symptoms of tetany. The signs of manifest tetany are mainly muscular spasms and convulsions. Certain groups of extremity muscles are most frequently affected, resulting in flexion of the upper and lower extremities, contraction of the hands, bending of the fingers, and downward bending of the toes. The muscles of the face are contracted, giving it a stiff and rigid appearance; very frequently the muscles of the larynx are involved, resulting in difficulty in respiration.

Much more frequent, however, than manifest tetany is latent tetany. The latter is characterized by hyperirritability of the peripheral nerves when tested with galvanic electricity or mechanical irritation (tapping) of certain peripheral nerve trunks. Contractions of the muscles do not occur spontaneously but can be produced by the above-mentioned tests. Fleischmann suggested latent tetany

as an explanation of those cases of enamel hypoplasia in which no history of manifest symptoms can be obtained.

In infants suffering from either manifest or latent tetany, the calcium content of the blood serum is below normal, although the blood phosphorus is normal. This decrease in the blood calcium causes a preponderance of potassium and sodium ions and, as a result, an increased irritability of the muscles. The parathyroid glands control and regulate the calcium metabolism of the body; their surgical removal is followed by a decrease in blood calcium and by tetany, which may be cured by the administration of the gland extract. The clinical and laboratory evidence concerning the relationship between tetany, parathyroid glands, and enamel formation seems to be rather conclusive: the disturbed function of the parathyroid glands causes a decrease in blood calcium, resulting in hyperirritability of the muscles and tetany. This same disturbance also interferes with the calcification of the forming enamel, causing defective enamel or enamel hypoplasia. Both convulsions and hypoplasia are the result of the lowered level of blood calcium.

Rickets is a deficiency disease in which the calcium and phosphorus metabolism is disturbed. Calcium and phosphorus deficiency and lack of vitamin D and sunshine are the main etiological factors. The blood calcium may be normal or subnormal, but the blood phosphorus is always low. The bones are poorly calcified, and there are large amounts of uncalcified osteoid substance in the skeleton. Consequently, the bones are soft, which results in the development of characteristic bone deformities, such as bowlegs and deformities of the skull, spine, and ribs. Rickets and tetany frequently occur together. Hess states that "from a clinical point of view it may be considered that practically all infants with signs of tetany have rickets to some degree." In many cases in which the clinical history is one of rickets only, manifest or latent tetany may have been present at some time.

It seems likely that enamel hypoplasia unaccompanied by severe disturbance of the dentin calcification indicates tetany as the main etiological factor, whereas poor calcification of the dentin, shortness of the roots, and delayed eruption are more suggestive of rickets.

Although they are of great etiological importance, rickets and tetany are not the only causes of enamel hypoplasia. Any severe illness during the first or second year of life (exanthematous fevers, such as scarlet fever and measles, whooping cough, bronchitis, pneumonia, severe gastro-intestinal disturbances, and general debility and underdevelopment) may disturb the calcium metabo-

lism enough to cause gross defects in the forming enamel. All these conditions are occasionally responsible for enamel hypoplasia, especially if they are superimposed on a rachitic process. The vulnerability of the ameloblasts apparently varies widely in different individuals. Some children have well-formed teeth in spite of severe illness during infancy; others have enamel hypoplasia although their medical history offers no satisfactory explanation for it. One of the difficulties in any study of this kind is that enamel hypoplasia of the permanent teeth is usually not recognized before some of the teeth have erupted, at the age of eight or nine years, and by then it is difficult, often impossible, to obtain an accurate medical history of the first and second years of life.

A few words should be said concerning the treatment and prevention of enamel hypoplasia. There is no treatment in the strict sense of the word, for once a tooth is hypoplastic, nothing can be done to improve the poor formation of the enamel. However, if the enamel of the anterior teeth is very poorly formed, they can be covered with porcelain jacket crowns with very satisfactory results. In the molars the defective occlusal surfaces can be built up by large inlays or overlays.

Prevention is of paramount importance. Since the calcification of the permanent teeth takes place entirely after birth, the responsibility for the prevention of enamel hypoplasia rests with the pediatrician. Careful and systematic health supervision, proper treatment of all diseases of infancy, correct nutrition, therapeutic and prophylactic administration of calcium, phosphorus, and vitamin D, and plenty of sunshine and fresh air can do much toward its prevention.

HISTOPATHOLOGY OF HYPOPLASTIC TEETH.

In order to understand the morbid anatomy of dental hypoplasia it is necessary to study not only ground sections through fully formed hypoplastic teeth but also tooth germs in which the defects can be examined in the various stages of development. Since human tissues of the latter type are naturally limited, many additional investigations have been carried out in the teeth of animals, in which hypoplastic changes can be produced at will in any degree and intensity, and the cellular phenomena studied afterwards.

Histopathology of Human Tooth Germs With Enamel Hypoplasia.—In 1920 Gottlieb reported the histological findings in decalcified jaws of infants in which the forming tooth germs showed the

type of changes that lead to enamel hypoplasia. He studied five jaws, ranging in age from two months to one and one-half years. One of these tooth germs is shown in Figure 22. The distribution of the enamel, which has stained dark purple with hematoxylin, is very irregular. In some areas the enamel is apparently of normal thickness; in others, it is very thin or missing, exposing the underlying dentin. The enamel surface is uneven, with deep pits or grooves on the labial and lingual sides of the crown.

The enamel epithelium has undergone extensive degeneration; it has become detached from the enamel surface, and the intervening space contains cells and cell detritus. The ameloblasts have disappeared; the stratum intermedium and the outer enamel epithelium show irregular proliferation. Under these circumstances, no further enamel formation can take place, and had the child from whom this specimen was obtained lived and the tooth erupted, it would have resembled that illustrated in Figure 27. The dentin of this tooth germ shows evidence of very poor calcification, namely, a wide layer of uncalcified dentin matrix and numerous interglobular spaces.

Another type of enamel hypoplasia is illustrated in Figure 23 in the tip of a permanent incisor of an infant that died with symptoms of severe rickets. Near the incisal edge two layers of enamel can be distinguished: next to the dentin a thick layer of normal enamel, and near the surface a thin layer that stained a much deeper shade of purple. Near the incisal edge the outer zone of enamel is completely missing, thus exposing the deeper enamel; the enamel epithelium over the latter has been reduced to a few scattered cells, whereas the rest of the surface is covered with normal ameloblasts. It can be assumed that in this case the enamel formation took place

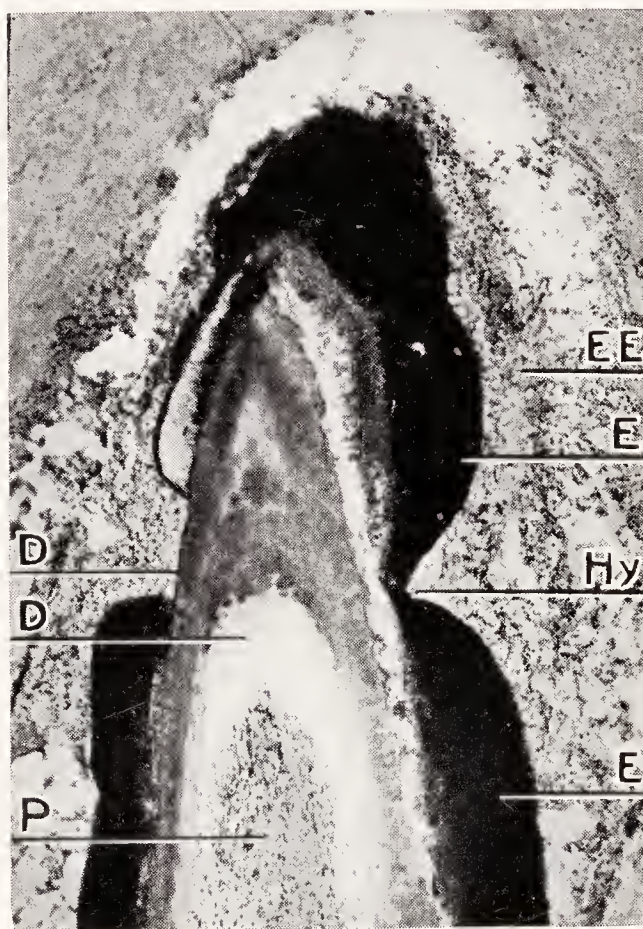


FIG. 22.—Tip of a permanent tooth germ of a child, aged one and one-half years, with severe enamel hypoplasia. The enamel epithelium, *EE*, is detached from the enamel and shows extensive cell proliferation. *E*, poorly calcified enamel; *Hy*, enamel hypoplasia in a corresponding level on the labial and lingual side of the germ; *D*, exposed dentin at the bottom of the hypoplasia; *D'*, wide layer of predentin; *P*, pulp. (Gottlieb, courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)

normally at first. Later the infant became ill, and from then on the enamel calcified poorly and in some areas failed to form.

The mechanism of the formation of enamel hypoplasia has been explained in two ways. Neither explanation alone can completely account for all the tissue changes, and in the author's opinion there are still several phases of the problem that need clarification.

One explanation of enamel hypoplasia is based primarily upon the physical characteristics of young enamel (Gottlieb). Like any other hard substance, enamel is laid down in an uncalcified form, as a kind of matrix which later becomes calcified. The amount of this matrix is very small, and normal enamel formation is possible

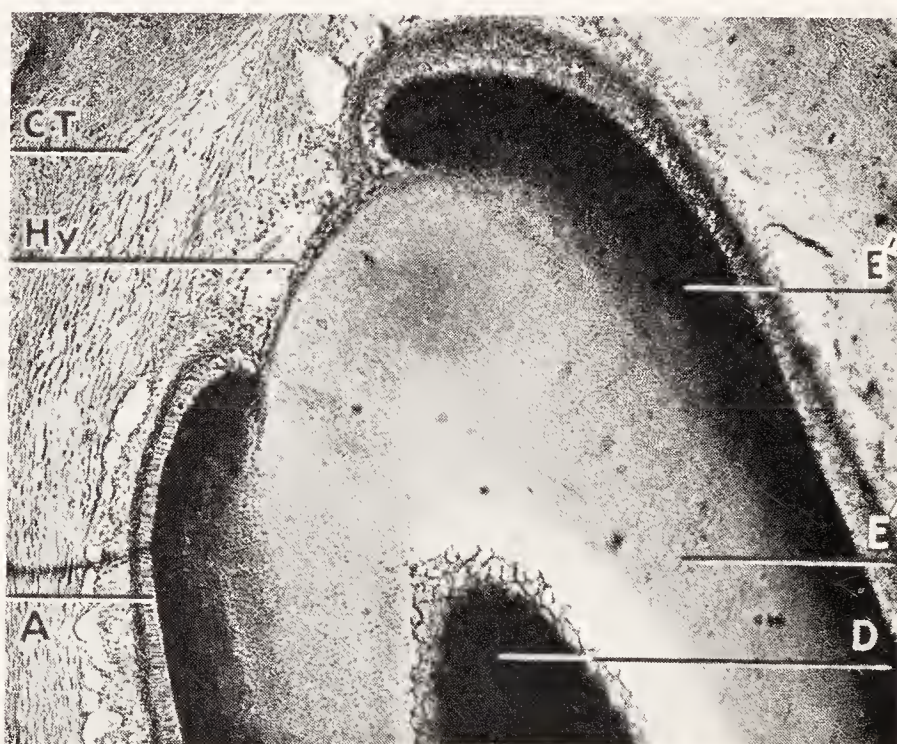


FIG. 23.—Enamel hypoplasia near the incisal edge of a permanent tooth germ of a rachitic child. *D*, dentin; *E'*, inner zone of enamel; *E''*, outer zone of enamel; *A*, ameloblasts; *Hy*, enamel hypoplasia; *CT*, connective tissue surrounding the germ. (Courtesy of J. R. Blayney.)

only if calcification follows immediately after its formation. If however, the calcification of the matrix is delayed, the latter is unable to keep its shape; it folds, collapses, as it were, which folding later forms the irregular grooves and depressions on the enamel surface. Even though this collapsed matrix later becomes calcified, the damage to the configuration of the enamel surface is irreparable.

One of the strongest arguments in favor of this “collapse theory” is the presence of lines of injury (Zsigmondy's *Unterbrechungslinien*) in adult hypoplastic teeth. These lines run from the bottom of the hypoplastic defects to the dentino-enamel junction (see Fig. 26). They resemble very pronounced stripes of Retzius, but whereas the enamel rods run continuously and undisturbed through the Retzius

lines, they are interrupted at these lines of injury. Often the Retzius lines on the outer side of a line of injury converge toward one point, as if the thickness of the enamel had actually been reduced by a collapse of its layers.

The lines of injury can be compared to the faults in geology. A fault is a section of rock layers that has slipped, so that there is a break in the continuity of the strata. Similarly the enamel rods have slipped along a line of injury, and the rods adjacent to the break are often warped and distorted.

The other explanation of enamel hypoplasia deals primarily with the degenerative changes in the ameloblasts (Bauer, Klein). In the

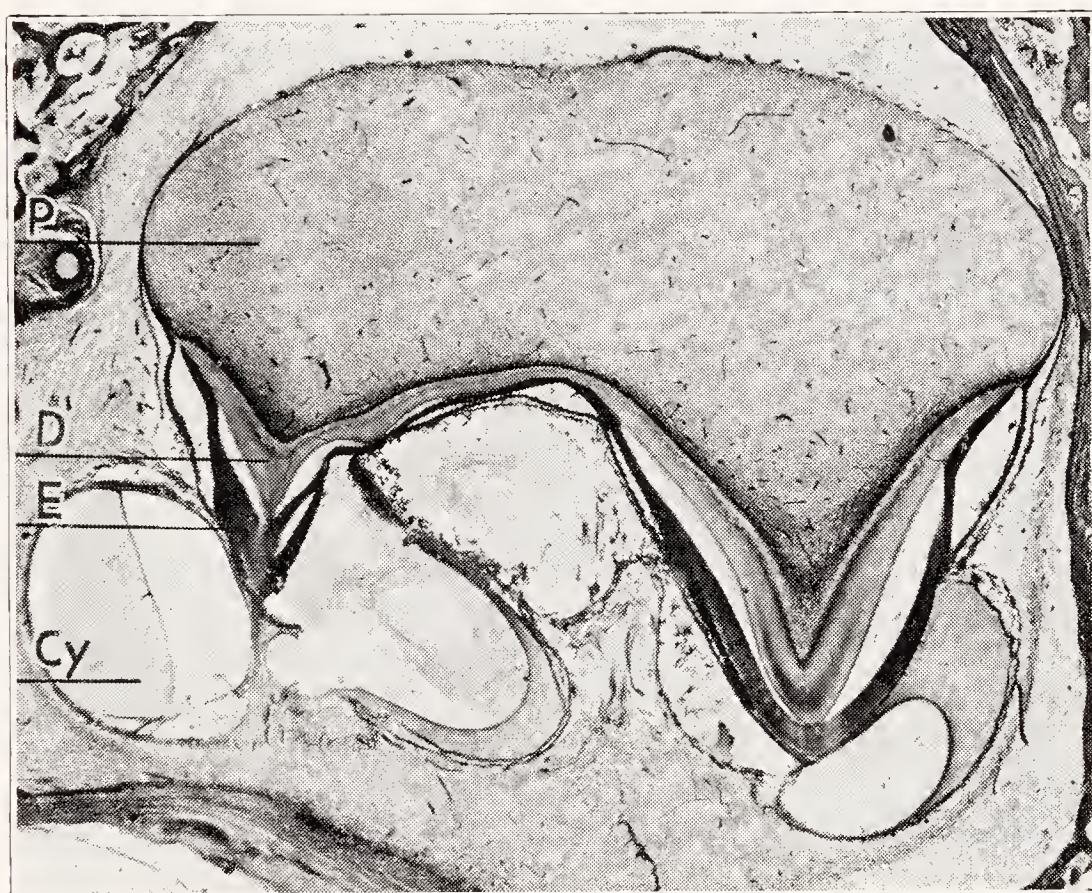


FIG. 24.—Germ of upper first permanent molar of a child, aged nine months, with history of severe rickets. *Cy*, cystic degeneration of enamel organ; *E*, enamel; *D*, dentin; *P*, pulp. (Kronfeld, Jour. Am. Dent. Assn.)

germs of hypoplastic teeth the ameloblasts are found in all stages of degeneration, from slight irregularities and vacuolization to complete destruction; likewise the other layers of the enamel organ are affected. Frequently these changes begin with an edema of the ameloblasts and an accumulation of fluid between ameloblastic layer and enamel surface. The enamel epithelium becomes detached from the forming enamel, and cystic cavities form in the outer layers of the tooth germ (Fig. 24). In the areas where this cystic degeneration of the enamel epithelium has taken place, no more enamel is laid down. The depth of the hypoplastic defect depends upon the

amount of enamel present at the time when the ameloblasts begin to degenerate: if the disturbance occurs very early in the process of amelogenesis, no enamel at all may be formed; if it occurs later, the bottom of the hypoplastic area is covered with a layer of enamel of varying thickness.

Boyle reported atrophy and degeneration of the ameloblasts and cessation of enamel formation in the tooth germs of a three and one-

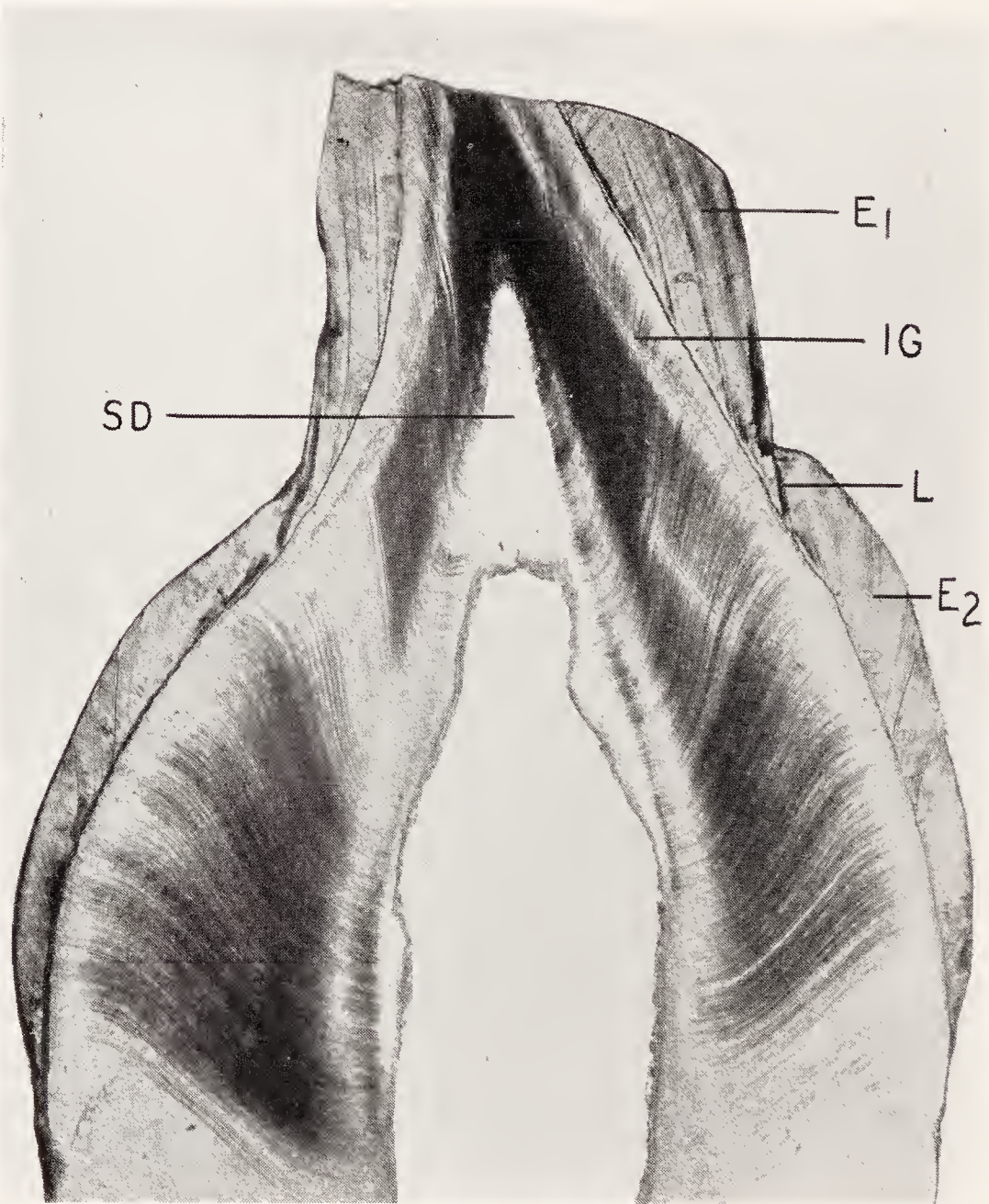


FIG. 25.—Ground section through lower central incisor with enamel hypoplasia. *E*₁, enamel laid down before the hypoplasia occurred; *L*, line of injury; *E*₂, enamel laid down after the hypoplasia occurred; *IG*, interglobular spaces corresponding to the enamel hypoplasia; *SD*, secondary dentin due to abrasion.

half months old infant with a clinical history of vitamin A deficiency. The dentin of these tooth germs was defectively calcified. From these findings it can be concluded that vitamin A deficiency may occasionally be the cause of hypoplasia in human teeth.

Histopathology of Completely Developed Human Teeth With Enamel Hypoplasia.—The distribution and structure of enamel

and dentin in adult hypoplastic teeth can best be studied in ground sections. Figure 25 shows a lower incisor with a single hypoplastic defect in the enamel. At the bottom of the defect the lower portion of the enamel overlaps the upper; the two layers are separated by a definite line of injury. In the dentin there is a single layer of large interglobular spaces, corresponding chronologically to the line of injury in the enamel. From this specimen it can be concluded that the individual suffered a single attack of a deficiency disease or

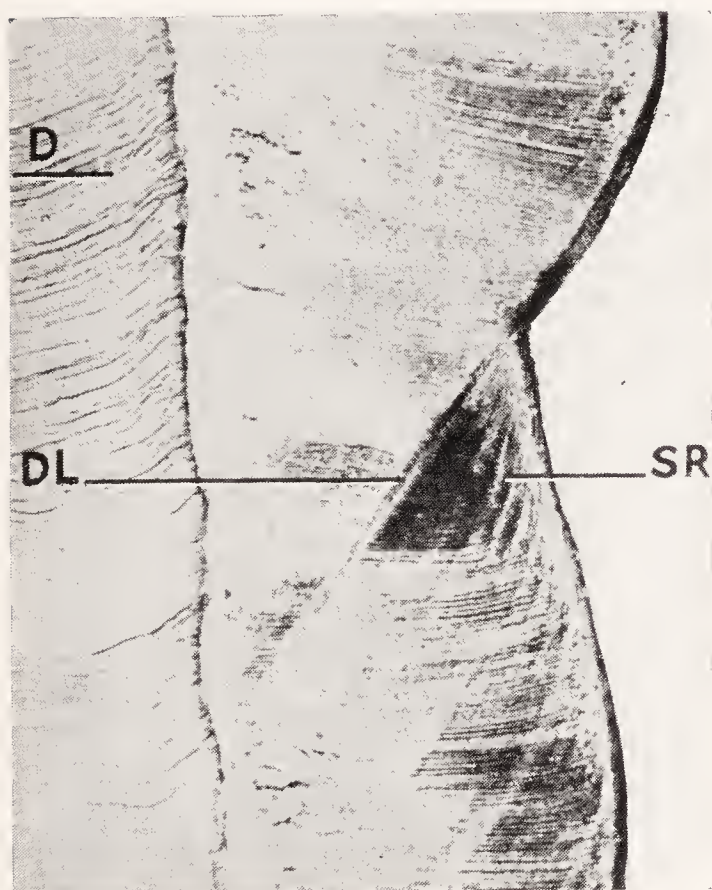


FIG. 26

FIG. 26.—Shallow enamel hypoplasia on a human incisor (ground section). From the bottom of the hypoplasia a line of injury, *DL*, extends into the underlying enamel. The enamel to the right of the line appears superimposed on that to the left. *SR*, stripes of Retzius converging toward the bottom of the hypoplasia; *D*, dentin.

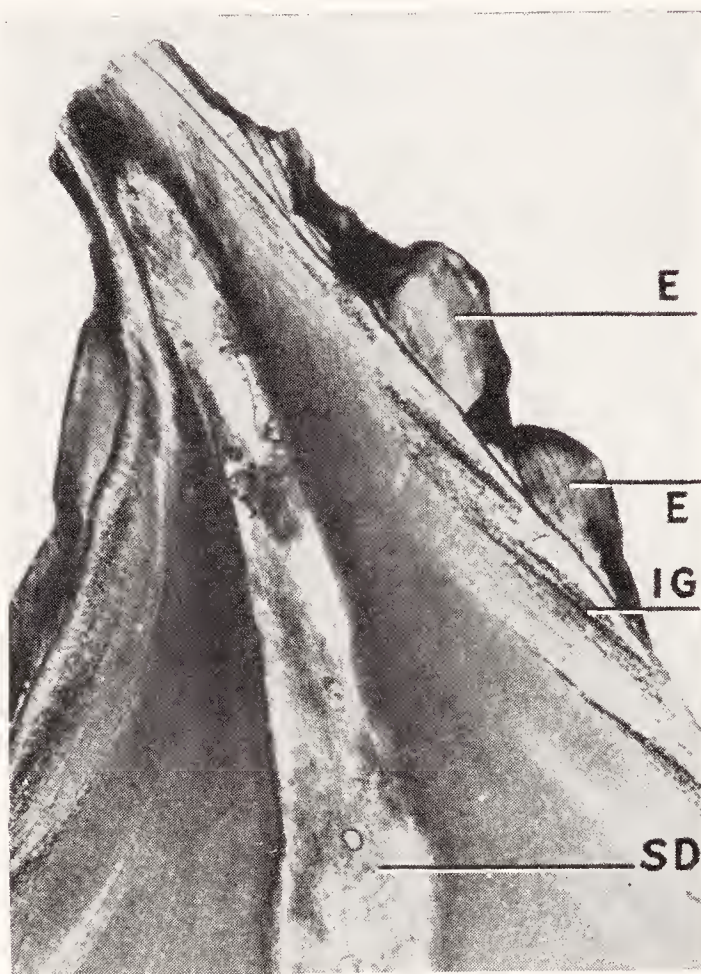


FIG. 27

FIG. 27.—Labio-lingual ground section through an incisor with severe enamel hypoplasia. On the labial surface of the crown the enamel is arranged in several horizontal ridges, *E*, between which the dentin is exposed. *IG*, interglobular spaces in the dentin arranged in rows corresponding to the distribution of the enamel hypoplasias; *SD*, secondary dentin in the pulp chamber. (Courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)

other illness at the age of approximately two years; before and after that time normal enamel and dentin were laid down.

The line of injury and the arrangement of the enamel rods and Retzius lines on either side of it are shown in Figure 26, taken from another tooth with a similar defect.

A tooth with severe hypoplasia in which deep defects alternate with ridges of poorly formed enamel is shown in Figure 27. A

separate layer of interglobular spaces in the dentin corresponds to each hypoplastic zone in the enamel.

ENAMEL HYPOPLASIA AND DENTAL CARIES.

Contrary to general belief, enamel hypoplasia *per se* is not an important etiological factor in dental caries. It is true, however, that hypoplastic teeth decay more rapidly than normal teeth, once decay has started. If an individual is immune to caries, intact as well as hypoplastic teeth will remain free of caries; if he is susceptible, then the teeth will decay regardless of whether they were intact or hypoplastic, but the destruction will spread more rapidly and extensively in hypoplastic teeth.

The above statements are confirmed by statistical data. Calteux made an extensive study of enamel hypoplasia and caries in all grade school children of the Grand Duchy of Luxemburg. He found hypoplastic teeth in 8.45 per cent of all children. Of the hypoplastic first permanent molars 88 per cent were carious; the corresponding figure for non-hypoplastic first permanent molars was 68 per cent. Thus, it becomes apparent that although nearly 90 per cent of all hypoplastic molars decayed, this percentage is only about 20 per cent higher than that for decay in non-hypoplastic molars among children living under similar external conditions. For the anterior teeth this difference is still smaller.

Shelling and Anderson have reported similar findings. They observed enamel hypoplasia of the permanent teeth in 25 per cent of a group of children with a clinical history of rickets and in 10 per cent of a group with no clinical record of rickets. The caries incidence in the rachitic group was not greater than in the non-rachitic group. From their observations Shelling and Anderson concluded that no relationship exists between rickets and dental caries, and that vitamin D cannot be considered a protection against caries of the deciduous teeth.

Other investigators have come to different conclusions. Melanby found a much higher incidence of caries in hypoplastic deciduous teeth than in non-hypoplastic ones. Eliot, *et al.* compared the incidence of caries in non-hypoplastic and hypoplastic permanent teeth. Of the former 36 per cent were carious; of the latter, 50 per cent.

As yet there is no satisfactory explanation for the discrepancy between these various reports. Perhaps there are racial and geographical factors involved, since some of the investigations were carried out in different countries, and in some both Negro and white children were examined. Also there was quite a variation in the

ages of the children, a factor which may have affected the figures for the incidence of caries, although it would not have altered those of hypoplasia.

Because of pits and other surface irregularities on normally immune tooth surfaces, caries in hypoplastic teeth occurs in atypical places, such as on the labial surface of the anterior teeth or on the tips of the cusps. For this reason, and also because of the poor quality of the surrounding tooth structure, it is often quite difficult to restore the lost tooth structure satisfactorily.

ENAMEL HYPOPLASIA OF LOCAL ORIGIN (TURNER TEETH).

Occasionally hypoplastic enamel is observed that is confined to single teeth, especially bicuspid. The affected teeth, known as Turner teeth, have small, brown, irregularly-shaped crowns; the enamel may be entirely lacking or may be present in zones or islands in some portions of the crown.



FIG. 28



FIG. 29

FIG. 28.—Radiograph of the first and second deciduous lower molars of a child, aged five and one-half years. Destruction of crown and periapical infection in the second deciduous molar. (Bauer, *Ztschr. f. Stom.*, courtesy of Urban & Schwarzenberg.)

FIG. 29.—Section through area shown in Fig. 28. The enamel epithelium of the second bicuspid has been destroyed by the periapical inflammation of the second deciduous molar, and its enamel is hypoplastic. (Bauer, *Ztschr. f. Stom.*, courtesy of Urban & Schwarzenberg.)

Obviously this form of hypoplasia cannot be caused by a systemic disease or deficiency, for if it were, other tooth groups would also be affected. The cause of enamel hypoplasia on single teeth is infection and apical inflammation of the preceding deciduous teeth.

The pathology of this process has been described by Bauer and by Morningstar. Figures 28 and 29 show two of Bauer's illustrations. The pulp of a lower deciduous molar has become infected and necrotic. Chronic periapical inflammation has developed; it has spread to the enamel organ of the underlying tooth germ and has damaged the ameloblasts of the latter.

The possibility of such damage to the crown of a developing permanent tooth by an infection of its predecessor is an additional argument for the early care and treatment of the deciduous teeth.

DISTURBED CALCIFICATION OF THE DENTIN.

Dentin formation normally occurs in the following way: After the dentin matrix has been formed by the pulp tissue, calcium salts



FIG. 30.—Interglobular spaces in the dentin. Ground section. The areas of predentin are surrounded by calcified dentin and bordered by the spherical surfaces of the globules. *IG*, interglobular spaces.

are deposited into it. Normally the precipitation of these salts takes place to within a short distance of the pulp; thus the latter is lined by only a very thin layer of uncalcified dentin matrix (predentin, dentinoid). The lime salts are deposited in small, round globules which grow by concentric deposition and finally unite to form the fully calcified dentin.

If the calcification of the dentin is disturbed, not all the globules of calcification unite; areas of matrix remain uncalcified, surrounded by convex calcified globules. These areas are called interglobular spaces or, preferably, interglobular dentin (Fig. 30), since they are

not true spaces but the uncalcified dentin matrix between the globules. If dentin calcification is severely disturbed, the interglobular spaces are numerous and large. In severe rickets, the dentin may be entirely uncalcified except for a few small calcified globules. The latter condition has been produced experimentally in the incisors of white rats by feeding them a diet deficient in vitamin D. As a result of disturbance of the calcium metabolism, the dentin is very poorly calcified. Figure 31 shows a portion of the labial surface of an upper incisor of a rat that had been kept on a rickets-producing

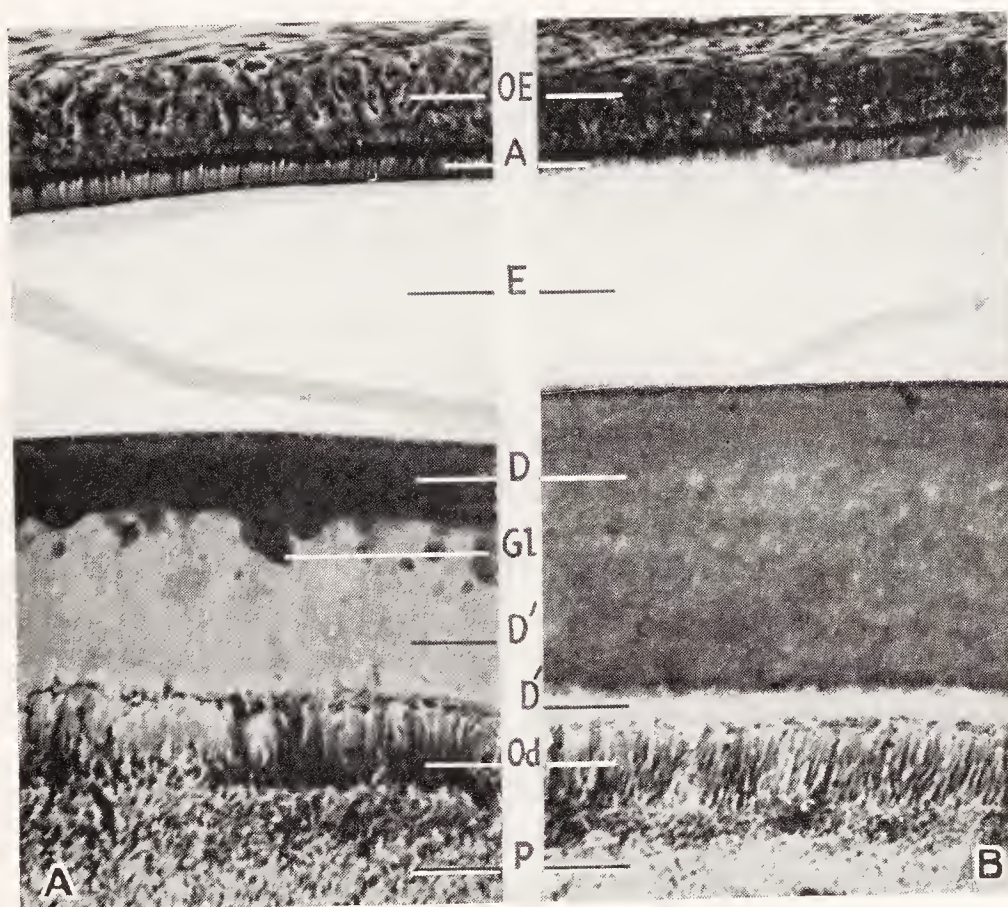


FIG. 31.—*A*, Incisor of a rat fed for eight weeks on a diet deficient in vitamin D. Poor calcification of the dentin; wide layer of uncalcified dentin matrix. *B*, corresponding area of an incisor of a control animal, fed on a balanced standard diet. Normal calcification of the dentin. *OE*, outer enamel epithelium; *A*, ameloblasts; *E*, enamel; *D*, calcified dentin; *Gl*, globules of calcification; *D'*, dentinoid (uncalcified dentin matrix); *Od*, odontoblasts; *P*, pulp. (Kronfeld and Barker, Jour. Am. Dent. Assn.)

diet for eight weeks. Only the layer of dentin directly beneath the enamel is calcified; the next layer is dentin matrix with irregularly scattered globules of calcification; and the innermost layer, most recently deposited, shows no calcification, for it consists entirely of matrix. In order to make these changes plainer, a section through the corresponding area of the incisor of a control animal is reproduced alongside the other. The dentin of the control animal is well calcified throughout; only next to the pulp chamber is there a layer of predentin.

Defective calcification of the dentin, either without or with accompanying hypoplasia of the enamel, has also been produced

experimentally in the teeth of animals by feeding them diets deficient in vitamins A and C (Wolbach and Howe). The addition of these vitamins to the diets resulted in repair and improved calcification. With the exception of the observation by Boyle (see page 48) no correlation has as yet been made between these experimental findings in animals and observations of dental deficiencies in man.

Changes very similar to those produced experimentally in animals are found in the dentin of individuals with rickets. Figure 32

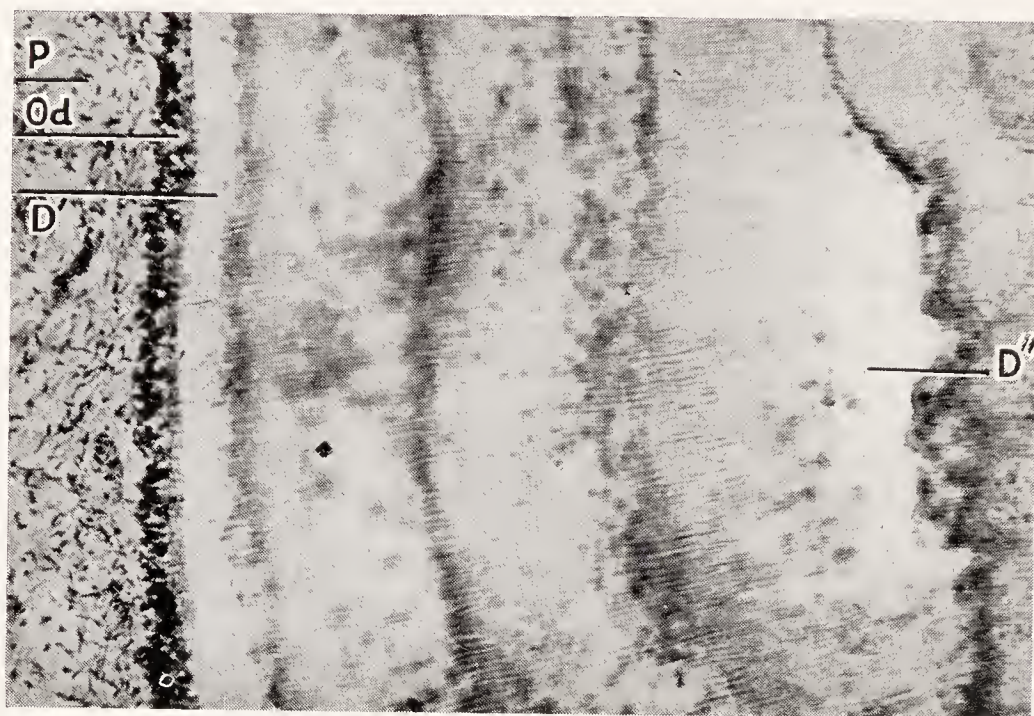


FIG. 32.—Rachitic dentin in a permanent molar of a child. Zones of very poor dentin calcification alternate with better calcified layers. *P*, pulp; *Od*, odontoblasts; *D'*, uncalcified dentin matrix next to the pulp; *D''*, matrix without globules. The dark zones indicate the presence of globules of calcification. The light zones consist of uncalcified matrix.

shows the dentin of a human first permanent molar. Bands or zones of very poor calcification alternate with zones of somewhat better calcification. In some places the matrix contains no calcium globules. Next to the pulp chamber is a wide zone of dentinoid. A higher magnification of this area shows the great irregularity of the border between calcified and uncalcified dentin (Fig. 33). In one place the odontoblasts have been destroyed by a capillary hemorrhage. At PC a group of pulp cells has been embedded in the dentin matrix. The latter phenomenon, the inclosure of cells or capillaries in the matrix, is frequently observed in disturbed dentin calcification.

The large amount of uncalcified matrix in rachitic teeth is comparable to the wide borders of osteoid found in the bones of the rachitic skeleton. This uncalcified matrix is partly the result of

delayed calcification of the normally formed matrix and partly of hypertrophy of the matrix, comparable to the excess osteoid in rachitic bone.

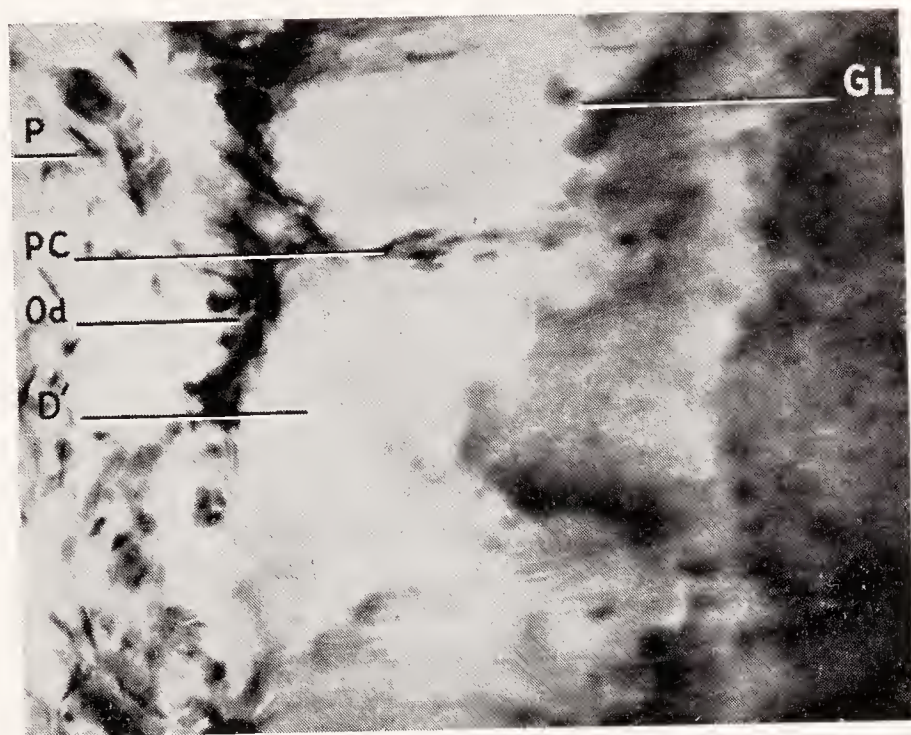


FIG. 33.—High magnification of odontoblasts and uncalcified dentin matrix in Fig. 32. *P*, pulp; *Od*, irregular odontoblastic layer; *D'*, uncalcified dentin matrix; *PC*, pulp cells enclosed in the uncalcified dentin matrix; *GL*, irregularly distributed globules of calcification.

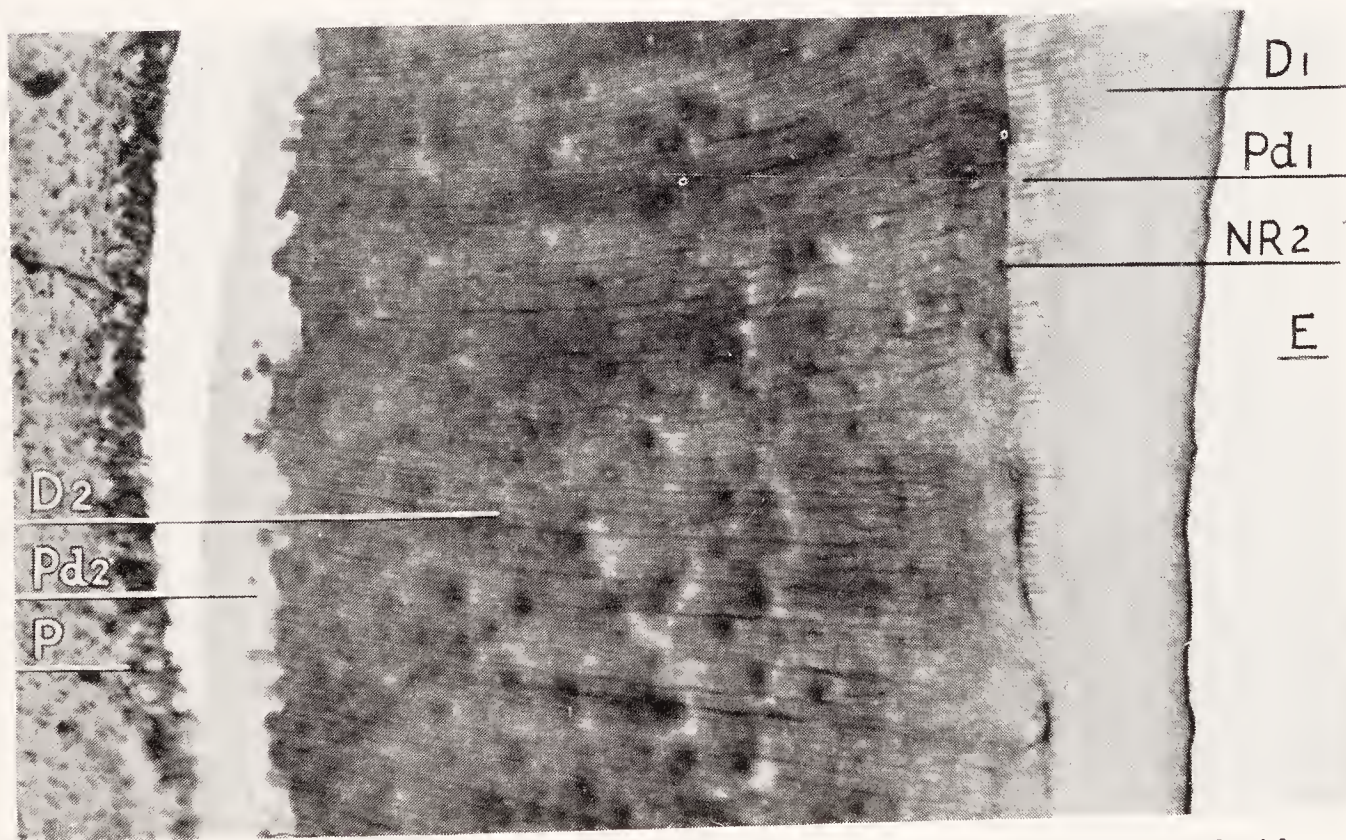


FIG. 34.—Decalcified stained section through the dentin of an upper first deciduous molar of a child with birth injury and neonatal dental hypoplasia. *E*, enamel space; *D*₁, prenatal dentin; *Pd*₁, prenatal predentin; *NR*₂, neonatal ring and plexus in the predentin; *D*₂, postnatal dentin; *Pd*₂, postnatal predentin; *P*, pulp. Note that the prenatal dentin is well and uniformly calcified, whereas the postnatal dentin is poorly calcified and shows many interglobular spaces. (Kronfeld and Schour, Jour. Am. Dent. Assn.)

The distribution of the interglobular spaces in poorly calcified dentin is not always the same. In some rachitic teeth they are arranged in distinct layers corresponding to the areas of hypoplastic enamel (Fig. 27); in others, especially in tooth germs, they are scattered throughout the dentin, apparently without relationship to the overlying enamel.

Figure 34 shows the dentin of the first deciduous molar of an infant that had been injured at birth. Two zones, divided by the neonatal ring, can be clearly distinguished in the dentin. The pre-

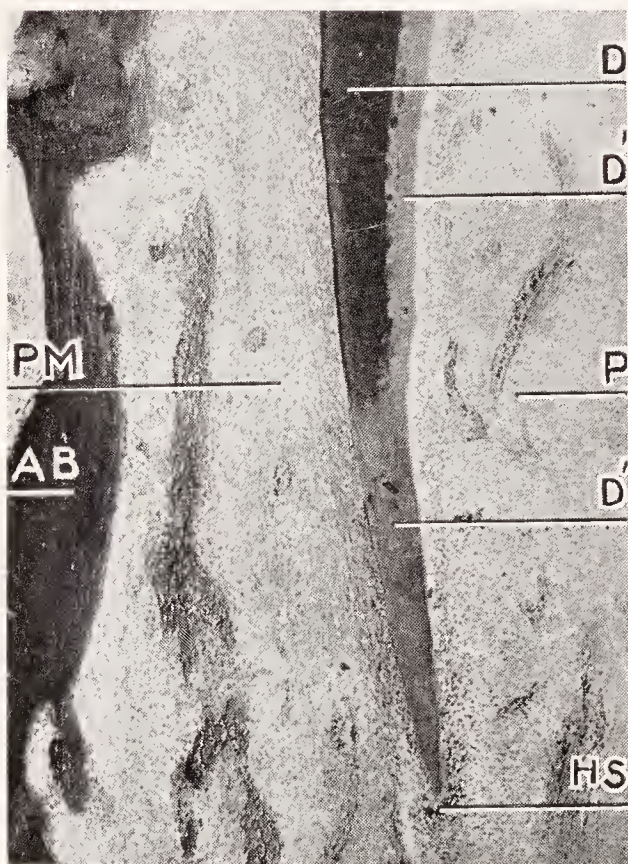


FIG. 35.—Root end of a deciduous molar of a rachitic child. Poor calcification of the dentin. The apex consists mostly of uncalcified dentin matrix. *D*, calcified dentin; *D'*, uncalcified dentin matrix; *HS*, Hertwig's epithelial sheath; *P*, pulp; *PM*, periodontal membrane; *AB*, alveolar bone. (Courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)

natally formed dentin is uniformly and well calcified; it contains only a small amount of interglobular dentin. The greater portion of the dentin, however, was laid down after birth; it is poorly calcified and abounds with interglobular spaces. The predentin adjacent to the pulp is abnormally thick.

In young teeth with developing roots the calcification of the root ends is retarded if the individual has rickets. Figure 35 shows the open root end of a deciduous tooth of a rachitic child. A considerable portion of the root from Hertwig's sheath crownward is uncalcified. The beginning of calcification is indicated by the darker staining of the dentin as compared with the matrix.

A basic difference between hypoplastic enamel and poorly calcified dentin is that enamel hypoplasia is irreparable, whereas poor calcification of the dentin can be improved, at least to some degree, by additional precipitation of lime salts. This has been shown in animal experiments, and most likely it also occurs in man. This secondary calcification of the dentin is probably the explanation for the defects in the enamel in many hypoplastic teeth being far more severe than those in the dentin.

In severe rickets the formation of dentin matrix without subsequent calcification does not go on indefinitely. At first, matrix

is formed as usual, but if no calcification takes place, matrix formation gradually slows down and finally ceases entirely. As a result, the teeth of individuals who have suffered from severe rickets often have shorter roots than average normal teeth.

An important clinical symptom in rickets is the delayed eruption of the teeth. This phenomenon can easily be duplicated in animal experiments. The incisors of rachitic rats grow more slowly than those of normal control rats; Orban found that practically any kind of dietary deficiency retards this rate of eruption. Bauer reported that in rachitic puppies eruption of the permanent teeth was considerably delayed as compared to control animals on a normal diet.

Hess tabulated the time of eruption of the first human tooth and found a marked delay in rachitic children; even in mild cases of rickets, the eruption is noticeably delayed. This delayed eruption in children cannot yet be fully explained. Knowledge of the mechanism of tooth eruption is incomplete, and although some of the component factors are well known, there is still no universally accepted and applicable answer to the question as to exactly what makes a tooth erupt. Several of the endocrine glands are involved, for subnormal function of the pituitary gland and the thyroid results in a definite retardation (Schour). But whether or how these facts are related to the delayed eruption in rickets is still being investigated.

HUTCHINSON'S TEETH.

Hutchinson's teeth are a congenital dental anomaly that consists of a hypoplasia of the upper and lower permanent incisors and of the permanent molars.

Typical Hutchinson's incisors have the following characteristics: The sides of the crown converge toward the incisal edge, so that the crown is wider in the cervical portion than near the incisal edge. The mesial and distal sides of the crown bulge slightly, and the incisal edge has a crescent-shaped notch (Fig. 36). The crown of such incisors has been compared to a screw-driver. Its greatest mesio-distal width is not at the level of the contact point but near the cemento-enamel junction.



FIG. 36.—Hutchinson's teeth. (Stumpf, Dent. Cosmos.)

Such incisors are often short and widely spaced. The teeth most often affected are the upper central incisors and the lower central and lateral incisors. The upper lateral incisors are frequently congenitally missing.

If the first permanent molars are involved, the crowns are short and contracted toward the occlusal surfaces. Because the enamel



FIG. 37.—Hutchinson's molars. (Karnosh, Arch. Derm. Syph.)

is arranged in small irregular globules, such teeth have been called mulberry molars (Fig. 37).

The mechanism of the formation of Hutchinson's incisors has been explained by de Jonge-Cohen. Normally an upper central

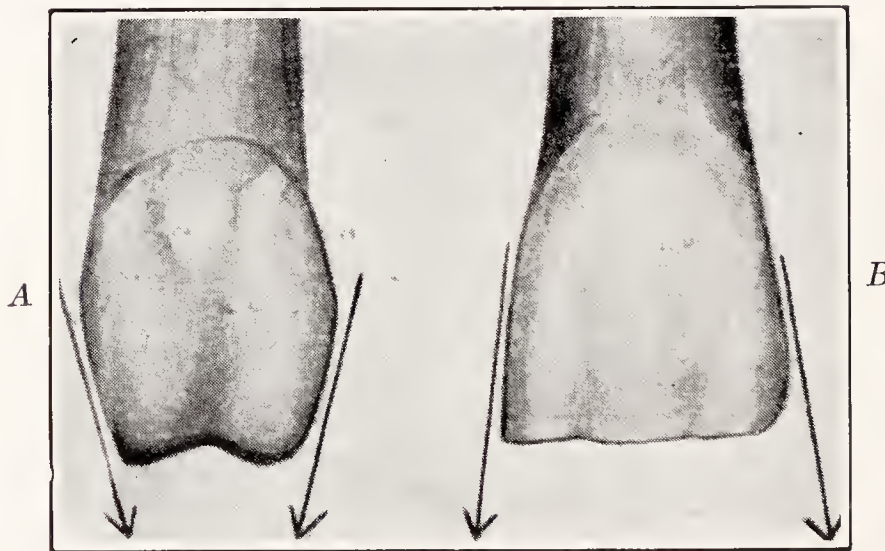


FIG. 38.—A, Hutchinsonian crown of an upper central incisor; B, normal crown of an upper central incisor. (Prinz and Greenbaum.)

incisor develops from three calcification centers, namely, a mesial, a central, and a distal marginal tubercle; these three tubercles are still visible at the time of eruption. In a Hutchinson's incisor, the central tubercle is missing; consequently, the mesial and distal tubercles are close together, with a semilunar notch between them (Fig. 38).

The semilunar notch of the incisors is the result of a developmental disturbance, not of wear or erosion. It sometimes happens, though, that Hutchinson's incisors erupt with a thin, atrophic ledge of enamel and dentin stretching across the notch, which is soon worn off and leaves behind the typical semilunar incision. Otherwise the enamel of such teeth is normal and smooth, unless there are hypoplastic defects, which may obscure the characteristic markings.

Such teeth are named after Jonathan Hutchinson, an English surgeon who, in 1857, described three conditions as diagnostic symptoms of hereditary syphilis. These three conditions, which are spoken of as Hutchinson's triad, are: inflammation of the eye, inflammation of the inner ear, and hypoplastic teeth.

Lesions of the eye are observed in the majority of patients suffering from hereditary syphilis. There is a chronic inflammation of the deep layers of the cornea (diffuse interstitial keratitis); the cornea becomes opaque, and blood-vessels appear in it. The condition may be progressive and cause loss of vision due to opacity of the cornea; in other cases the keratitis may heal, leaving merely fine, diffuse scars in the cornea.

The symptoms in the ear are inflammation of the inner ear (labyrinthitis) and of the middle ear (purulent otitis media). The condition is usually bilateral, and in many instances leads progressively to deafness.

Hutchinson's teeth are observed in about one-half of all heredosyphilitic patients. Abnormally small teeth, malocclusion, delayed eruption, and congenital absence of teeth are also frequently found in such individuals.

Ever since Hutchinson's first publication on the subject, the relationship between Hutchinson's teeth and hereditary syphilis has been the subject of much controversy and differences of opinion. Some investigators consider Hutchinson's teeth almost an infallible symptom of heredosyphilis; others class them with other forms of enamel hypoplasia and believe that they are due to rickets and to developmental disturbances, but that they are not a symptom of hereditary syphilis.

Cavallaro and Stein were among the early investigators who extensively studied the dentition in hereditary syphilis. Karnosh presented a chronological analysis of Hutchinson's teeth; he clearly distinguished between the typical deformity associated with hereditary syphilis and other hypoplasias of nutritional origin that may be superimposed upon Hutchinson's teeth and may produce a composite lesion.

The histological examination of the teeth and jaws of syphilitic

infants by Boyle, Pflüger, and Burkett revealed inflammatory and degenerative changes in the developing tooth germs that can well explain the abnormal structure of Hutchinson's teeth. Pflüger's specimens showed perivascular round cell infiltration in the pulp tissue and degeneration of the enamel epithelium. In the permanent central incisor germs, the atrophy of the enamel organ was distributed in such a way as to explain the formation of Hutchinson's incisors. Burkett described degeneration, edema, and cyst formation in the enamel epithelium of the first permanent molars and lack of enamel formation on some of the cusps.

Some investigators claim to have demonstrated *Spirocheta pallida* (*Treponema pallidum*) in sections through the tooth germs of still-born syphilitic infants. Others are of the opinion that there is no final evidence of spirochetes in the dental anlagen of syphilitic infants (Hill).

The present-day opinion is that true Hutchinson's teeth are highly suggestive of hereditary syphilis. Occasionally, in non-syphilitic individuals, hypoplastic defects resembling Hutchinson's teeth are observed, which are the result of nutritional and metabolic disturbances in early childhood. Therefore, the clinical diagnosis of hereditary syphilis can be made only if, in addition to Hutchinson's teeth, there are other symptoms of Hutchinson's triad present, and if the serological reaction indicates a syphilitic infection.

MOTTLED ENAMEL.

Mottled enamel is enamel that is poorly calcified and discolored as a result of disturbances during its formation. Usually only the permanent teeth are affected, although mottled enamel has been observed in the deciduous dentition (Smith and Smith). In teeth only slightly affected, the enamel has a dull, opaque, chalky-white appearance (Fig. 39); in those more affected, there is a yellowish or light brown discoloration, and in those most severely affected, the enamel may be dark brown or almost black. In many instances, all of these variations are found in one and the same tooth: normal enamel, chalky areas in which the normal translucency is absent, yellow discoloration, and dark brown bands or stripes (Fig. 40).

The areas most commonly mottled are the labial surfaces of the upper and lower central incisors, lateral incisors, and cuspids; the lingual surfaces are involved to a lesser degree. Occasionally, in severe cases, the enamel of all the teeth is mottled to some extent.

The surface of mottled teeth is usually smooth and even, and with the finest explorer no irregularities or defects can be detected. In

severe cases, however, the labial surface has rows of irregular shallow pits in it, in addition to the discoloration, similar to the condition in enamel hypoplasia.



FIG. 39.—Mottled teeth. The enamel is chalky white with brown bands. (Bunting.)

Mottled enamel is endemic in many parts of the world (Dean). In addition to its occurrence in the United States, it has been found in England, Italy, North Africa, Japan, China, and Argentina.

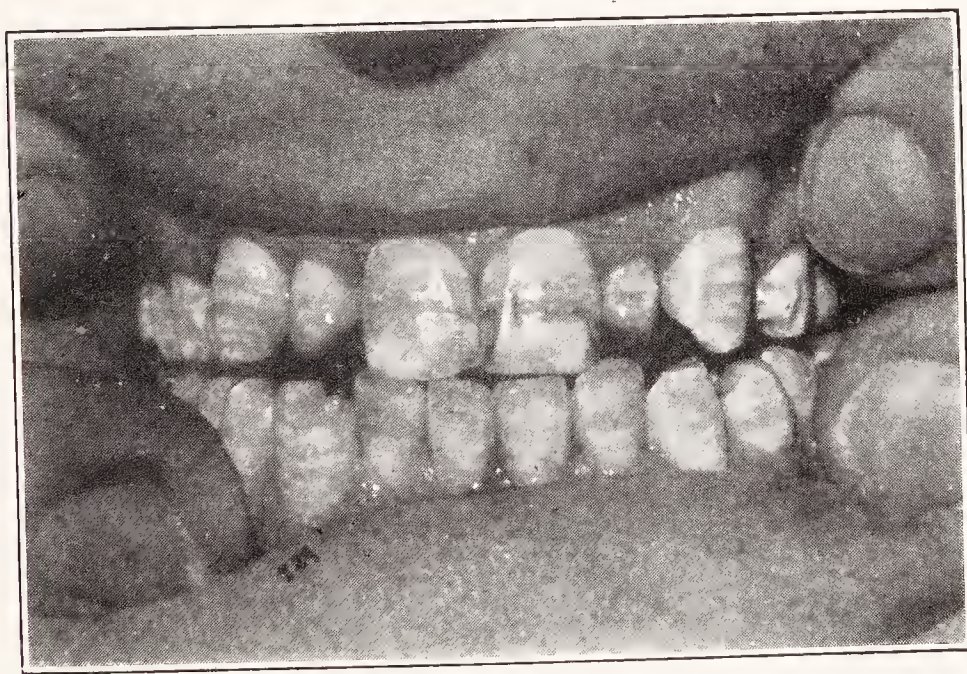


FIG. 40.—Mottled teeth. Brown discoloration of the enamel. (Bunting.)

A varying percentage of people living in any particular district are affected, regardless of race, color, or sex. Dean, who has studied the distribution of mottled enamel over the entire United States, reported in 1938 about 345 areas in which it is endemic. Eighty-six per cent of these areas are located west of the Mississippi River,

94 of them in the State of Texas. Other states in which mottled enamel is frequent enough to constitute an important public health problem are Colorado, South Dakota, and Arizona. There are also a few scattered endemic areas in Illinois, Idaho, Virginia, the Carolinas, and other states (Fig. 41).

The histopathology of mottled teeth has been investigated by Black and McKay, Williams, Applebaum, and others. The enamel

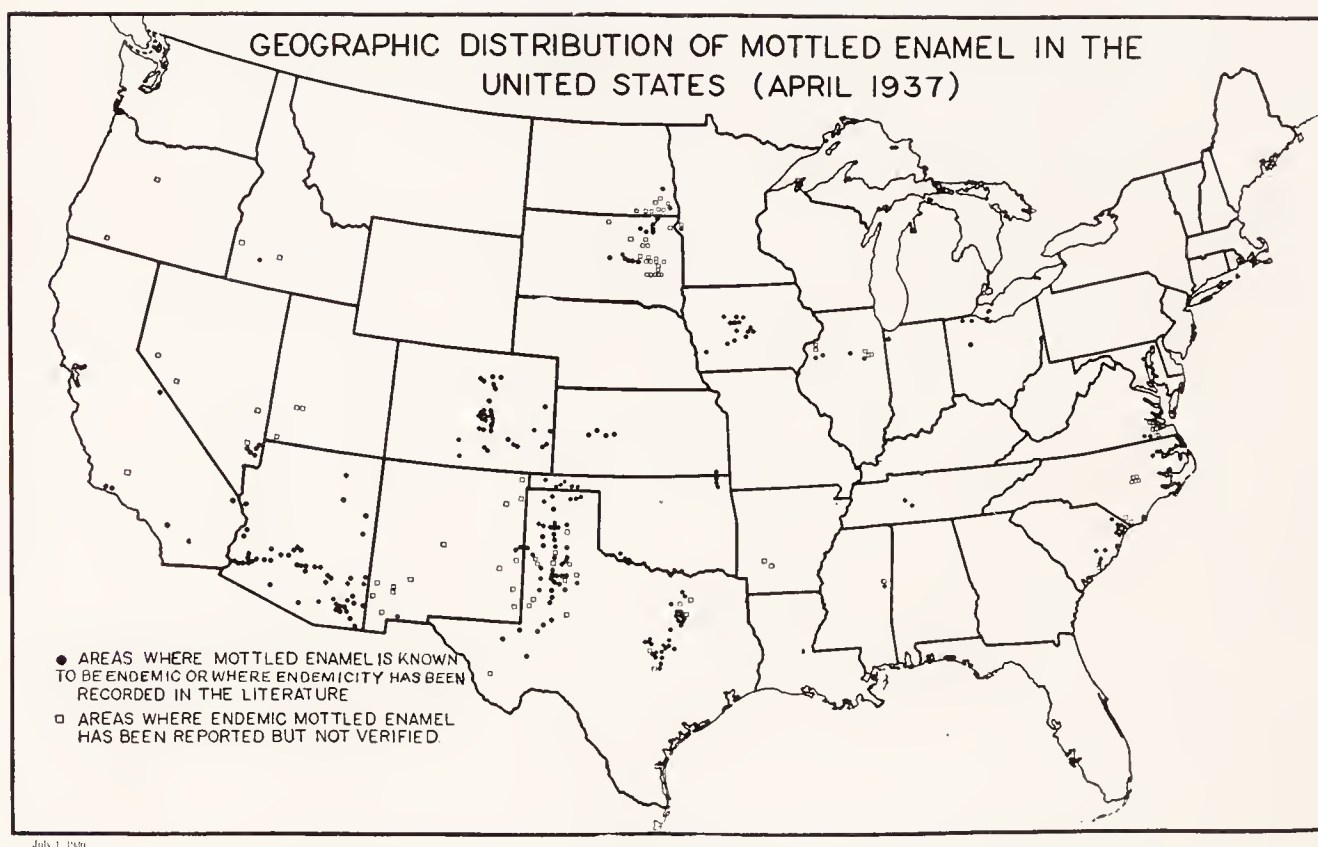


FIG. 41¹

structure is defective, the enamel rods are incompletely calcified, and the interprismatic substance is poorly developed or absent. A brown pigment is present between the enamel rods. In spite of this defective structure, the incidence of caries in mottled teeth is not greater than in normal teeth.

The mottled condition of the teeth develops during the period of enamel formation; it is present when the teeth erupt, and it cannot be altered or influenced after eruption. The etiology of mottled enamel was unknown until about 1931 when exact water analyses revealed a striking coincidence between mottled enamel and fluorides in the drinking water. This observation was confirmed by numerous additional findings, and today it is generally accepted that fluorine in the drinking water causes mottled enamel. Moreover, mottled enamel can be produced experimentally in the teeth of laboratory animals by the feeding or injection of fluorine salts.

¹ Dean in Gordon's Dental Science and Dental Art, Philadelphia, Lea & Febiger, 1938.

Schour and Smith studied the influence of sodium fluoride injections upon the incisor teeth of rats. The incisors of these animals showed brown rings, each ring corresponding to a single injection. Histologically, the brown zones were areas of defective calcification of the enamel. In the dentin, corresponding alternating rings of normal and poor calcification were observed. A sharply outlined zone of disturbed calcification of the dentin corresponded to each injection of sodium fluoride. From these findings it appeared that fluorine in low concentration has a specific toxic effect upon the enamel-forming cells and also upon the calcification of the dentin.

Fluorine is contained in the soil in the form of soluble fluorides of calcium, sodium, and potassium. The concentration of fluorine in the drinking water of areas in which mottled enamel is endemic ranges from 2 to 13.7 parts per 1,000,000. The investigations of Dean have revealed a definite relationship between fluorine concentration and the severity of mottled enamel. In cities in which the water supply has a fluorine content of from 0.6 to 0.9 parts per 1,000,000, from 2.4 to 10.6 per cent of the children are affected, but only slightly. In cities where the water contains 1.5 to 3.9 parts per 1,000,000, the incidence of mottled enamel increases from 24.5 to 90.3 per cent, and some of the teeth are more severely affected. In four cities having a fluorine content in the drinking water of 4.0, 4.4, 5.7, and 8 parts per 1,000,000, the incidence of mottled enamel was 88.2, 97.8, 100, and 100 per cent, respectively, with a considerable number of severe cases among the more susceptible children.

The fluorine affects only the ameloblasts of those teeth whose enamel is being formed. Thus, a person who spent the first six or seven years of his life in a locality where the drinking water contained a toxic amount of fluorine is likely to have mottled enamel in the permanent teeth (except the third molars), even though he may have moved later into a fluorine-free area. Conversely, if a child is brought at the age of six or seven years from a fluorine-free region into a region where mottled enamel is prevalent, his teeth will not be mottled, since at that age all of the enamel (except in the third molars) is completely calcified.

There is no reliable treatment known for mottled enamel. Neither defective calcification nor discoloration can be changed by therapy. If the discoloration is very severe, it may be advisable to cover the teeth with porcelain jacket crowns.

Mottled enamel is a serious civic problem. Although mottled teeth are not detrimental to the health of the individual, they are

unsightly and disfiguring. Since the etiology of the condition has been established, it can now be prevented, if the use of fluorine-containing drinking water is avoided during childhood. This can sometimes be done by a change in the source of the water supply. An example of this was reported by McKay in regard to the town of Oakley, Idaho. Prior to 1925, all native children in this community had mottling of the teeth, varying in intensity from white spots to dark stains and pits. In 1925, the water supply was changed, and the spring that had been used during the past ten years was abandoned. In 1933, when the permanent teeth of the children born after 1925 had begun to erupt, it was found that none of these children had mottled teeth; the enamel was normal and formed a striking contrast to the badly discolored teeth of the older children. In the meantime the significance of fluorine had become known, and water analyses were made that revealed that the water formerly used, which had caused the mottled teeth, contained 6 parts per 1,000,000 of fluorine, whereas the new water supply contained less than one-half of 1 part per 1,000,000.

If a complete change of the water supply is not feasible, it may be possible to eliminate fluorines from the available drinking water by some form of chemical water treatment. Among other chemicals, magnesium oxide is effective for this purpose (Elvove).

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CHAPTER IV.

REGRESSIVE CHANGES IN DENTIN AND PULP.

BEFORE entering into the pathology of the dental tissues, a group of conditions will be discussed to which the term "regressive changes" is often applied. These conditions are formation of secondary or irregular dentin, sclerosis of the dentin, atrophy of the pulp tissue, and formation of calcified bodies in the pulp tissue.

These deviations from the normal structure of dentin and pulp sooner or later occur in every human tooth and, therefore, must be considered separately from such pathological conditions as caries or pulpitis. The regressive changes in dentin and pulp develop gradually without any clinical symptoms and without interfering with the life or the function of the tooth. Therefore, they may be considered an expression of the general aging of the organism, and also to a certain extent a defense mechanism against the many mechanical and other influences to which every tooth is subjected.

SECONDARY (IRREGULAR) DENTIN.

In a young tooth with an intact covering of enamel, dentin formation occurs in a regular, undisturbed manner; the dentinal tubules converge toward the pulp in a regular pattern. This type of dentin is called primary or regular dentin because it is formed in the early part of the life of every tooth and because of the regular course of the dentinal tubules.

Sooner or later, after the tooth has erupted and has come into occlusion, the enamel is worn through by mastication, and the dentin is exposed on the occlusal surface. This results in injury and irritation of the Tomes' fibers which are exposed in the abraded area. In response to this stimulation the pulp hurriedly forms a protective layer of calcified tissue over the central ends of the injured dentinal tubules. This hard substance is secondary dentin. The course of the dentinal tubules is much less regular in secondary than in primary dentin, and, therefore, it is called irregular dentin. Secondary dentin contains less organic substance than primary dentin and is less permeable; in this way it is capable of protecting the pulp from injury or irritation from without. Its hardness is slightly less than

that of primary dentin (Hodge). The borderline between the two types of dentin is usually easy to see in microscopic specimens.

Different factors lead to the formation of secondary dentin; all of it, however, appears the same microscopically, regardless of the cause.

The following causes are known to stimulate the formation of secondary dentin:

Abrasion (attrition, occlusal wear).

Erosion.

Dental caries.

Dental operations involving the dentin.

Fracture of the crown without exposure of the pulp.

Advancing age.

Abrasion and Secondary Dentin.—In a mouth with all of the teeth in normal alignment, the incisal edges and occlusal surfaces of all

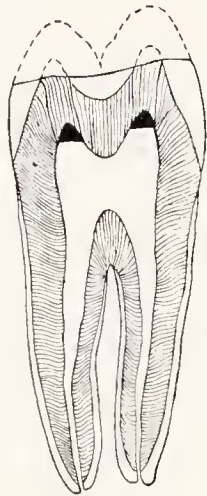


FIG. 42

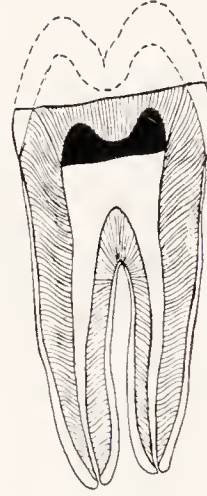


FIG. 43

FIGS. 42 and 43.—Diagrams of the distribution of secondary dentin in an upper bicuspid as the result of abrasion. The worn portion of enamel and dentin is indicated by dotted lines. The secondary dentin is indicated by black shading.

FIG. 42.—Beginning abrasion. The dentin is exposed at the tips of both cusps. Secondary dentin corresponding to the exposed area has formed in the pulp horns.

FIG. 43.—Advanced abrasion. A large portion of the dentin is exposed. The occlusal portion of the pulp chamber is filled by secondary dentin.

crowns are subjected to a great amount of wear. This is especially true of individuals with strong, sound teeth, for usually ten or fifteen years after the permanent teeth have come into occlusion, the enamel shows signs of considerable wear, with the formation of facets. In the fourth decade of life, as a rule, the dentin is exposed by abrasion, first at the incisal edge of the incisor teeth and the cusps of the molars. The process of wearing down continues until in older individuals with strong, healthy teeth the cusps have practically disappeared and dentin is exposed in almost every tooth, appearing as a yellow or brown area on the occlusal surface, surrounded by a white border of enamel (Figs. 42 and 43).

Teeth without cusps must be considered normal in old people. Man is equipped with one set of permanent teeth which are supposed to last a lifetime. Obviously these teeth cannot retain the shape they had at the time of eruption. The human teeth are much-used tools, and since there is no way of replacing the part of the crown that is lost by wear, the teeth, like most tools, will in time become worn down and shorter. This is known as physiological abrasion. If no pathological changes interfere, if no teeth are lost by caries or pyorrhea, abrasion will not become excessive before the natural limit of life is reached.

Unfortunately, in modern man physiological abrasion is a rare occurrence since malalignment, insufficient use, and caries usually preclude proper occlusal wear. It is almost necessary to go back to the teeth of primitive man to find what might be called the ideal form of abrasion: an intact set of thirty-two teeth with uniformly abraded cusps, lack of incisal overbite, and an occlusal plane which permits the lower jaw to move freely in every direction without cusp interference. This type of abrasion occurs so regularly in primitive man that anthropologists use the amount of occlusal wear found in a skull as an important criterion in determining the approximate age of the individual.

The best material on which to study physiological abrasion is prehistoric skulls. In the teeth of the North American Indians, for example, who lived centuries ago and whose skulls are found in the ancient burial grounds, all stages of abrasion from the early formation of enamel facets to complete absence of occlusal enamel can be studied. Abrasion occurred so regularly in these teeth that the differences in the degree of wear often correspond exactly to the sequence of eruption: in the same skull the first permanent molars may show the most extensive wear, the second molars less, and the third molars almost none.

Abrasion may also be pathological. Every dentist has seen older people with excessively abraded teeth, worn down almost to the gingivæ, sometimes with exposure of the pulps. This kind of pathological abrasion is the secondary result of another pre-existing pathological condition, such as loss of teeth, malalignment, or abnormal chewing habits; it is not comparable to ordinary, normal abrasion.

Pathological abrasion and excessive wear of some of the teeth are characteristic of certain occupations. Cobblers and seamstresses frequently have deep grooves in the anterior teeth which are caused by the habitual holding and biting of thread between the teeth. Glass blowers and musicians who play wind instruments suffer from

pathological abrasion of the anterior teeth, which are used to hold the mouthpieces of their instruments.

The amount of secondary dentin deposited depends upon several factors, namely, the extent of the exposed dentin area, the length of time that has passed since the dentin was exposed, and the individual reaction of the pulp. Some pulps seem to have a greater and others a lesser tendency toward the formation of secondary dentin.

Figure 44 shows a ground section through the crown of a lower

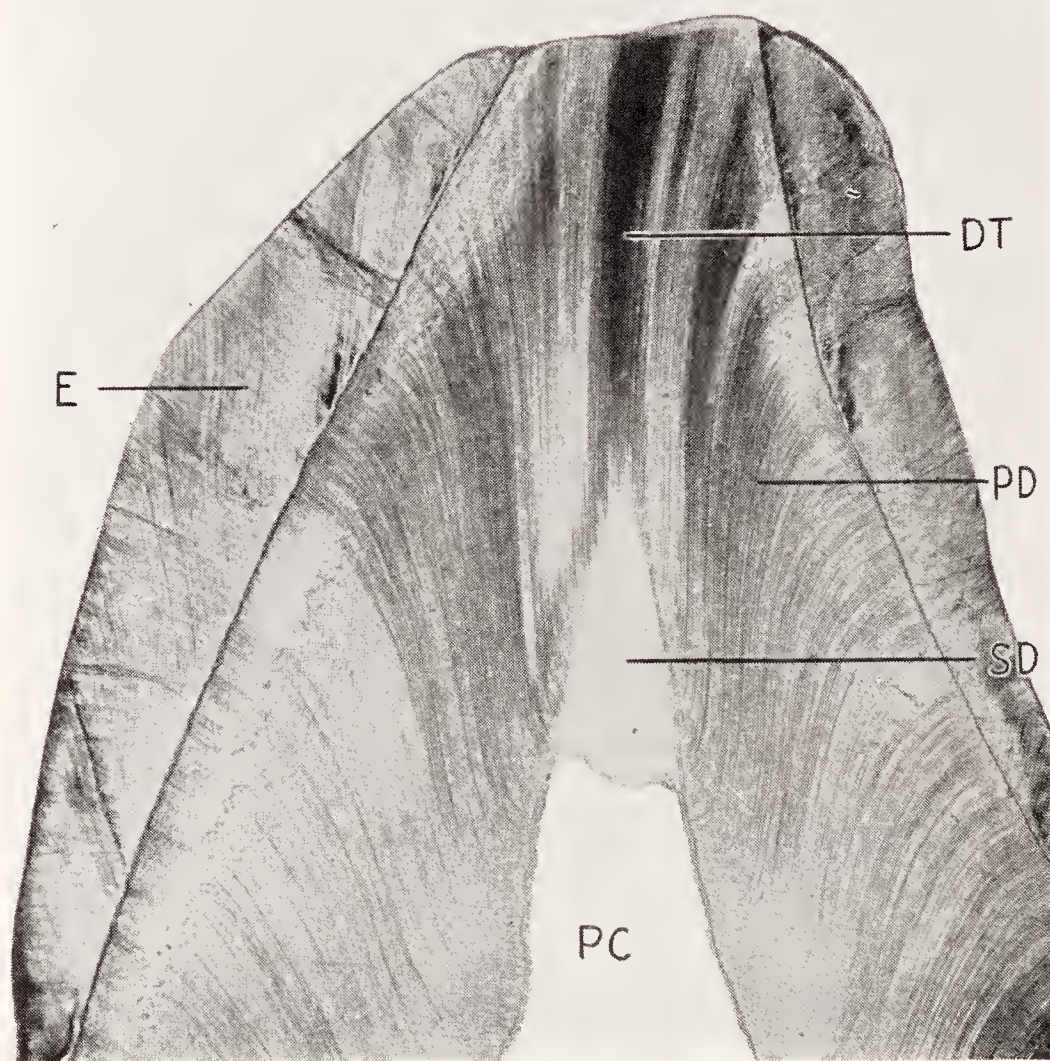


FIG. 44.—Ground section through the crown of an abraded lower cuspid. *E*, enamel; *PD*, primary dentin; *SD*, secondary dentin; *DT*, “dead tract” of exposed dentinal tubules; *PC*, pulp chamber.

cuspid with moderate abrasion that has exposed the dentin. The tip of the original pulp chamber has been filled by secondary dentin; the division between primary and secondary dentin can be easily recognized, since almost all dentinal tubules of the primary dentin end abruptly at this point and only very few continue into the secondary dentin.

As already mentioned, the main difference between primary and secondary dentin lies in the different course and number of the

dentinal tubules. This can be seen plainly in Figure 45, which was taken from a stained ground section. The regular, parallel dentinal tubules of the primary dentin end abruptly at the dividing line between the two kinds of dentin; from there on only a very few irregular tubules are present, running through the solid, calcified dentin matrix of the irregular dentin.

The formation of secondary dentin is especially important if abrasion reaches the level of the original pulp horns (Fig. 46). From this specimen it is evident that were it not for the secondary dentin, the pulp would have been exposed. As abrasion advances

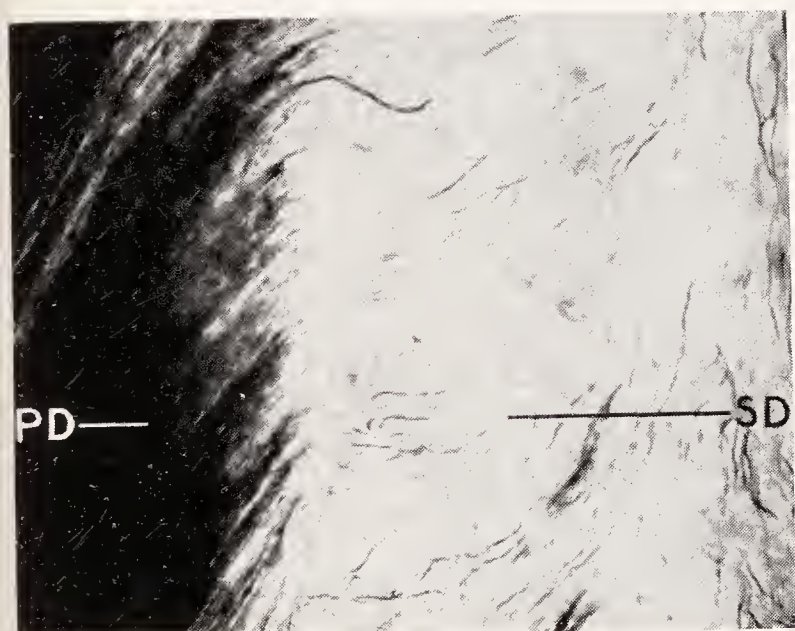


FIG. 45

FIG. 45.—Border between primary and secondary dentin. Ground section. *PD*, primary dentin; *SD*, secondary dentin. Note the small number and irregular course of the dentinal tubules in the secondary dentin. Most of the dentinal tubules of the primary dentin end at the border between primary and secondary dentin.

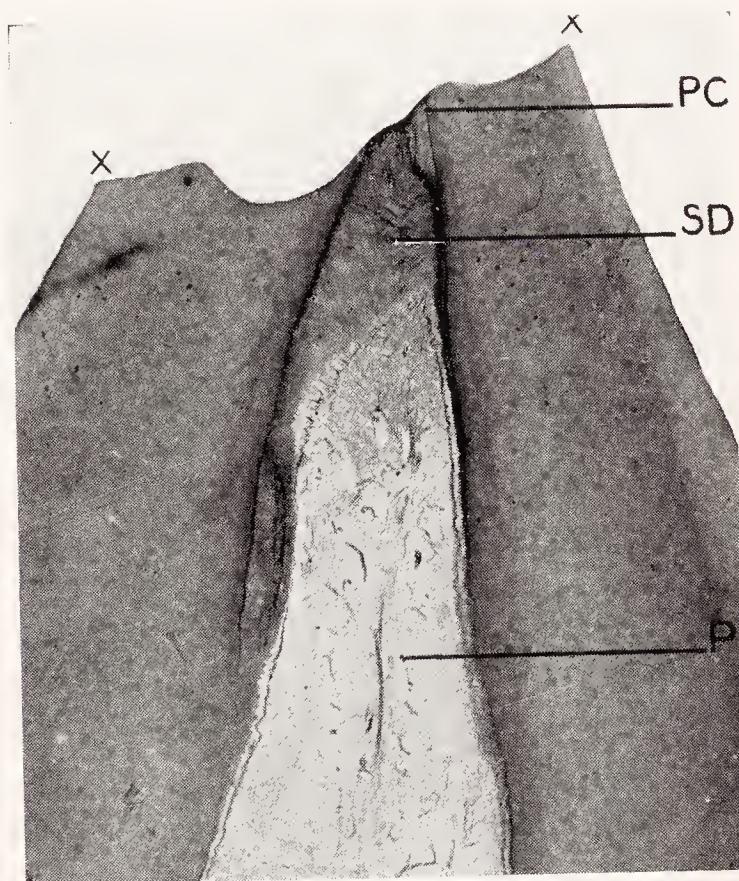


FIG. 46

FIG. 46.—Abrasion. Lower cuspid. At *PC* the dentin is worn down to the level of the original pulp chamber. *X-X*, abraded dentin surface; *PC*, outline of the original pulp chamber; *SD*, secondary dentin; *P*, pulp. (Courtesy of W. Willman.)

a considerable portion of the original pulp chamber that has been filled with secondary dentin may be worn away. But as long as this process of wearing down occurs slowly enough for secondary dentin to form, there is no danger of exposure of the pulp.

In old people sometimes almost the entire pulp chamber may be filled by secondary dentin (Fig. 47). This fact is important because such teeth do not necessarily respond to the vitality test; dental operations, such as grinding or drilling, usually painful in teeth with vital pulps, can be performed on such old teeth with little or no sensation.

The immediate stimulus causing the formation of secondary dentin seems to be the injury to the odontoblastic processes in the dentinal tubules. This opinion is supported by the observation that secondary dentin is formed only at the central endings of those dentinal tubules of which the periphery has been exposed. Fish regards the deposition of secondary dentin as a specific reaction of the dentinal tubules to injury. He called attention to the fact that two

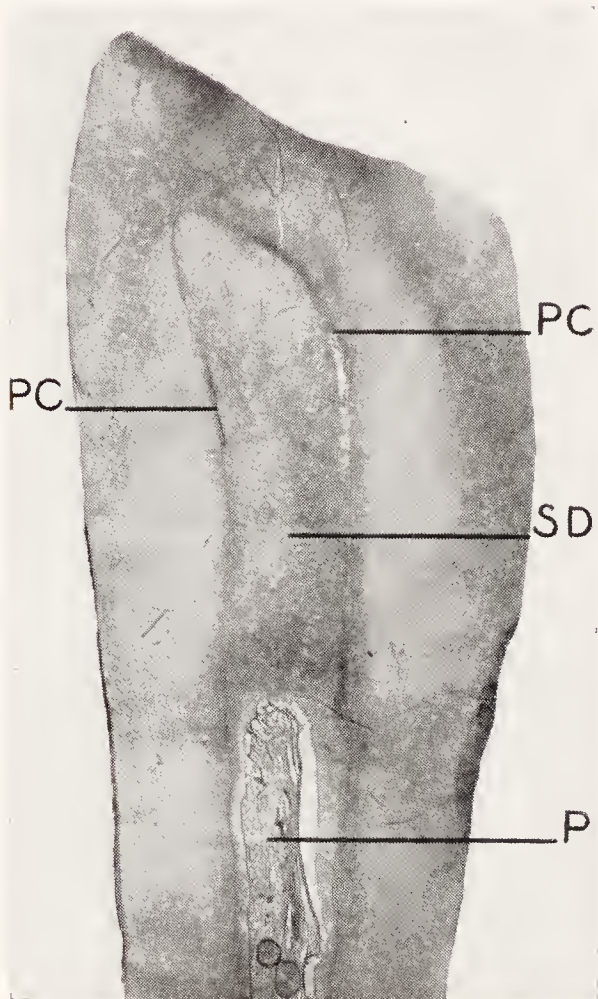


FIG. 47.—Advanced process of obliteration of the pulp chamber by secondary dentin. Lower incisor of an old individual. *PC*, original outline of pulp chamber; *SD*, secondary dentin; *P*, pulp. (Courtesy of W. Willman.)

phases can sometimes be distinguished in the formation of secondary dentin. Immediately following injury to the protoplasmic process, a barrier of calcium salts is deposited over the central opening of every injured tubule; then secondary dentin is formed on top of this barrier. The tubules in the primary and secondary dentin are thus completely separated by a calcified obstruction, and the primary dentin is cut off from the pulp. Clinically this is manifested by a loss of sensitiveness in the involved portion of the dentin. Fish calls the tract of dentin between the peripheral lesion and the secondary dentin a “dead tract.” He shows that if methyl blue dye is introduced into the pulp chamber of an extracted human tooth, the dye enters the dentinal tubules of the intact primary dentin and penetrates to the dentino-enamel junction, but wherever sec-

ondary dentin covers the central ends of the tubules, no dye can enter and the dentin remains unstained.

The pulp tissue under secondary dentin shows certain characteristic changes. As a result of the injury to Tomes' fibers, the odontoblastic layer may disappear and be covered by secondary dentin, in which event the new hard substance consists of a structureless, calcified mass without dentinal tubules. However, some of the odontoblasts usually survive, leaving their protoplasmic extensions tortuously and irregularly embedded in the secondary dentin. The number of odontoblasts is always greatly reduced; they are smaller than usual and their arrangement is irregular (Fig. 48).



FIG. 48.—Secondary dentin in a pulp horn of a lower molar caused by abrasion. On the surface of the secondary dentin the odontoblasts are small, irregular, and in places missing. *PD*, primary (regular) dentin; *SD*, secondary (irregular) dentin; *OD*, odontoblasts; *P*, pulp.



FIG. 49



FIG. 50



FIG. 51



FIG. 52

FIGS. 49 to 52.—Size of the pulp chamber. Comparison of labio-lingual sections through upper central incisors of different ages.

FIG. 49.—Age, eight years. Open root end; wide pulp chamber and root canal.

FIG. 50.—Age, fourteen years. Root fully formed. No abrasion; no secondary dentin formation.

FIG. 51.—Age, about thirty-five years. Incipient abrasion; secondary dentin in the incisal portion of the pulp chamber.

FIG. 52.—Age, about fifty-five years. Advanced abrasion. A large part of the original pulp chamber has become obliterated by secondary dentin. The root canal is very narrow.

As a result of the continuous formation of primary dentin and later of secondary dentin, the size of the pulp chamber of all human teeth slowly and steadily decreases with advancing age. In the anterior teeth the deposition of secondary dentin considerably reduces the length of the pulp chamber; at the same time the root canal becomes narrower. These changes will be illustrated by a comparison of labio-lingual sections through upper central incisors at different ages (Figs. 49 to 52). Figure 49 illustrates a section through an incisor shortly after eruption, at the age of eight years. The pulp chamber is very large; the root end is wide open. Figure 50 shows a section through the corresponding tooth of a boy, aged fourteen years. The root end is fully developed. The pulp chamber is smaller, but no incisal abrasion has yet taken place, and therefore, only primary dentin is present. In Figure 51 the corresponding section of an individual, aged thirty-five years, is reproduced. The process of abrasion has reached the dentin at the incisal edge; secondary dentin has been formed, considerably decreasing the size of both pulp chamber and root canal. In Figure 52 a corresponding tooth is illustrated from a man about fifty-five years of age. There is advanced abrasion; the crown has been worn flat. The pulp chamber has been reduced to a narrow space, and more than two-thirds of its original length has been obliterated by secondary dentin.

Erosion and Secondary Dentin.—Occlusal abrasion must not be confused with erosion. Erosion is a loss of tooth structure sometimes found on the labial or lingual side in the gingival third of a tooth. These defects are the result of a gradual wasting away of enamel and dentin without apparent cause. The most common form of erosion consists of wedge-shaped defects with sharply outlined borders; they are deeply cut into the tooth structure, and their floor is clean, hard, and smooth in appearance, altogether different from the usual appearance of dental caries (Fig. 53). On rare occasions, the enamel may be lost over most of the labial surface of the anterior teeth, resulting in shallow, dish-shaped eroded defects that extend into the dentin. Thus, the basic difference between abrasion and erosion can easily be recognized: abrasion is found only where opposing teeth come in contact, and its shape depends upon the amount of contact between the grinding tooth surfaces; erosion, on the other hand, is characteristically found on tooth surfaces which are not subjected to occlusal wear.

The etiology of erosion is unknown. Many theories have been elaborated to explain it; none as yet gives a satisfactory explanation. One of the supposed causes is vigorous use of the tooth brush in a

horizontal direction, together with a highly abrasive dentifrice. It is true that erosion-like defects have been produced experimentally on extracted teeth by the use of a machine which imitates the strokes of the tooth brush; yet, erosion has been observed in individuals who have never used a horizontal brushing motion, and even in some who have never brushed their teeth. Chemical processes have been cited as a possible cause, but so far convincing evidence is still lacking. Erosion has been found in individuals with acid as well as alkaline saliva. The differences between the saliva of people with



FIG. 53.—Erosion on the labial side of a lower cuspid (upper row) and first bicuspid (lower row) of a man, aged fifty-five years. Note the clean-cut, wedge-shaped defects that extend to the original pulp chambers. The latter are obliterated by secondary dentin.

and of those without erosions are not large or significant enough to permit any definite conclusions.

Just as uncertain as the etiology is the prognosis of erosion. Sometimes the process comes to a spontaneous standstill. Then fillings that have been inserted to replace the lost tooth structure may remain in place. Again, the wasting process may progress; then extensive destruction may result, and such a tooth may actually be cut in two by erosion. Occasionally erosion will progress around a filling that was inserted to replace the lost tooth structure, until the filling stands out like an island in the eroded area, and finally falls out.

The pulp reacts to erosion in the same way as it does to abrasion, namely, by the formation of secondary dentin. Because of this, a

wedge-shaped erosion may penetrate to the level of the original pulp chamber without exposing the pulp.

Dental Caries and Secondary Dentin.—When caries reaches the dentino-enamel junction it causes the formation of secondary dentin in the pulp chamber. By tracing the course of the dentinal tubules from the carious lesions to the pulp, it can be seen that the secondary dentin corresponds in extent and locality to the decayed area. The lack of tubules in the secondary dentin may be considered a



FIG. 54.—Secondary dentin formation under caries. Mesio-distal section through lower molar with occlusal (*OC*) and mesial (*MC*) caries. *SD'*, secondary dentin corresponding to the occlusal decay; *SD''*, secondary dentin corresponding to the mesial decay. The dentinal tubules leading from the mesial decay to the pulp chamber converge toward the pulp; therefore, the area of secondary dentin formation is smaller than the carious area of the dentin surface. (Coolidge, Illinois Dent. Jour.)

protective measure against the possible invasion of the pulp by bacteria.

In Figure 54 is shown a mesio-distal section through a lower molar with both occlusal and mesial caries. There are two separate deposits of secondary dentin on the walls of the pulp chamber, one on the roof, which corresponds to the occlusal caries, and another on the mesial wall. Several calcifications (pulp stones) can be seen in the pulp tissue.

The combined influence of abrasion and caries produces a condition like that illustrated in Figure 55. In a lower incisor with mesial and distal caries, secondary dentin has formed on the mesial and distal walls of the pulp chamber; rootward the secondary dentin

ends abruptly. From here the dentinal tubules extend upward and reach the dentin surface at the apical end of the decayed area. There is secondary dentin at the incisal end, as well as on the walls, of the pulp chamber; this has evidently been caused by the incisal abrasion that was present in this tooth. The original partition of the incisal end of the pulp chamber into three horns, corresponding

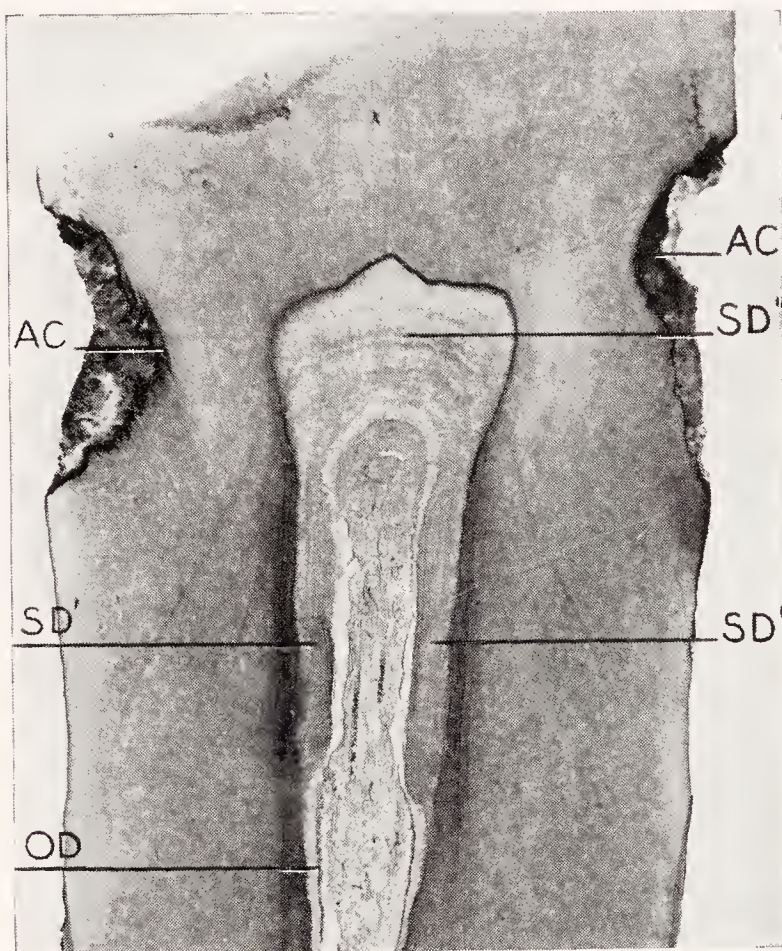


FIG. 55.—Secondary dentin under caries and abrasion. Mesio-distal section through lower incisor. *AC*, approximal caries; *SD'*, secondary dentin corresponding to the approximal caries; *SD''*, secondary dentin corresponding to the incisal abrasion; *OD*, odontoblasts rootward from the secondary dentin. (Coolidge, Illinois Dent. Jour.)

to the three small incisal tubercles of the erupting tooth, is still visible and marks the borderline between primary and secondary dentin.

It might be appropriate at this point to discuss the general relationship between caries and secondary dentin formation. In the specimens illustrated so far, the pulp responded successfully to approaching caries by the formation of secondary dentin. This is corroborated clinically by the observation of teeth with advanced caries in which, below the soft, decayed dentin, the pulp is still intact and well protected by a layer of secondary dentin. On the other hand, caries often reaches the pulp despite secondary dentin formation. Two factors play an important rôle here, namely, the rate of speed with which caries progresses and the individual reaction

of the pulp. So far as the first factor is concerned, it must be kept in mind that it takes anywhere from a few weeks to several months for the formation of secondary dentin of considerable thickness. Therefore, if the decay is very rapid, as, for instance, fissure caries in the first permanent molars of children, the pulp does not have time to form sufficient secondary dentin to ward off the rapidly spreading infection and decomposition of the dentin. If, on the

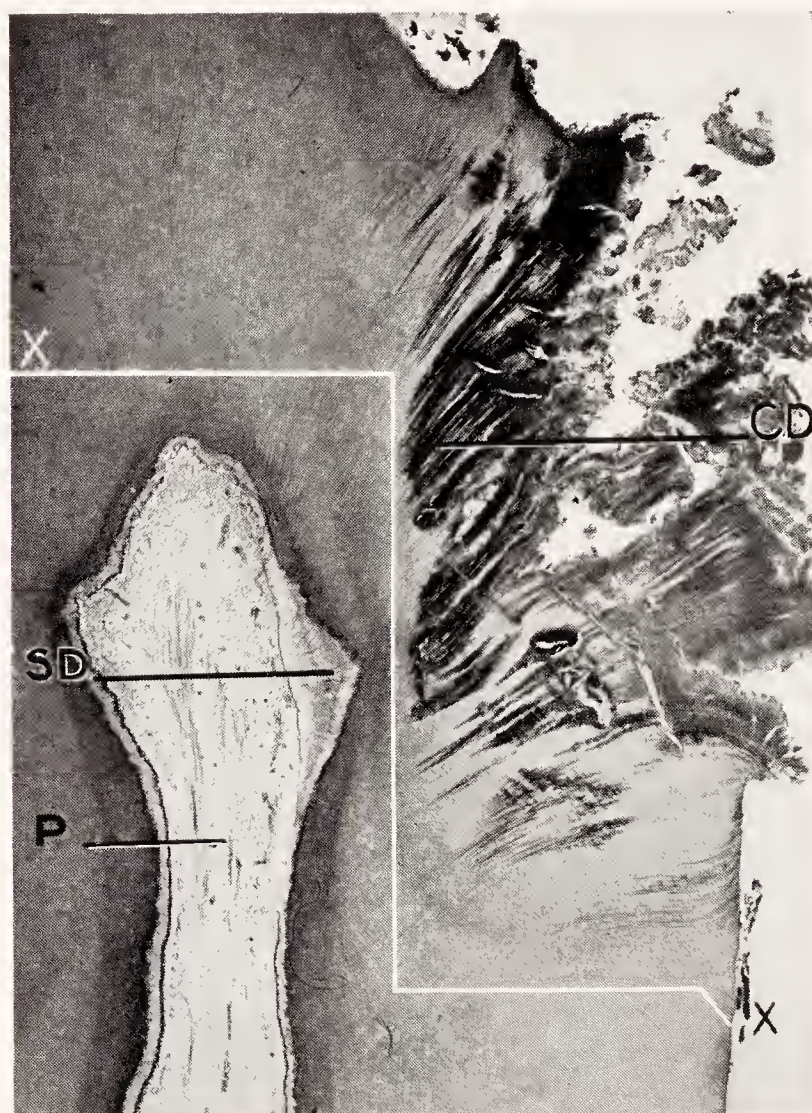


FIG. 56.—Caries on the distal side of a lower bicuspid. Deposition of secondary dentin on the distal wall of the pulp chamber. *CD*, carious, decomposed dentin; *SD*, secondary dentin; *P*, pulp. The pulp tissue is normal. The line *X-X* indicates the approximate outline of a cavity preparation that would remove all decayed dentin and leave a layer of dentin of uniform thickness, sufficient to protect the pulp.

other hand, the progress of caries is slow, secondary dentin forms as fast as the decay advances, and the pulp remains intact.

The second factor involved, namely, the individual reaction of the pulp, is not yet understood. Why one pulp forms a large amount of secondary dentin and, as a result, remains intact, while another shows only a weak defensive reaction and soon becomes infected or exposed, is one of the many problems of biology that are still awaiting solution. There is no doubt but that one pulp has greater resistance than another, but the factors that determine or regulate these individual variations are unknown.

Dental Operations and Secondary Dentin.—Every drilling or grinding of a tooth causes the formation of secondary dentin, provided the operation involves the dentin. The extent and amount of the deposition will depend upon the size and depth of the cavity, the age of the individual, the time that has elapsed since the operation was performed, and the individual reaction of the pulp. The larger the area of dentin that is exposed, the more secondary dentin will be deposited; the younger the individual, the more rapidly will the pulp react with such a deposition; and, finally, the more responsive the individual pulp to irritating stimuli, the more extensively will secondary dentin develop.

Before a cavity is prepared, a certain amount of secondary dentin is usually present as the result of caries (Fig. 56); however, since it is necessary to enlarge the cavity to comply with the rules of extension, new areas of dentin with intact dentinal tubules are cut by bur or stone, and secondary dentin is subsequently formed at the central ends of these freshly exposed tubules. If a large cavity is prepared in an intact tooth for the purpose of using the latter for a bridge abutment, no secondary dentin may be present at the time of operation.

The distribution of secondary dentin under an approximo-occlusal metal filling in a lower molar is illustrated diagrammatically in Figure 57. The area of the pulp chamber occupied by the secondary dentin is determined by the extent of the exposed dentin surface; the thickness of the secondary dentin is determined by the length of time that has elapsed since the filling was inserted and by the individual pulp reaction.

Another dental operation that causes secondary dentin formation is the grinding of a tooth with a stone. If, for instance, cusps or incisal edges of teeth are ground off enough to expose the dentin, the result will be exactly the same as in tooth abrasion. The only difference is that abraded teeth are usually not sensitive, whereas ground teeth are often very sensitive to thermal changes. During the comparatively slow process of abrasion, the sensitiveness of the exposed tubules is reduced by the gradual calcification at their proximal ends. In grinding, however, vital tubules are suddenly exposed on the dentin surface, and, as a result, the dentin is sensitive until, within the next few weeks or months, secondary dentin seals the central ends of the injured tubules.

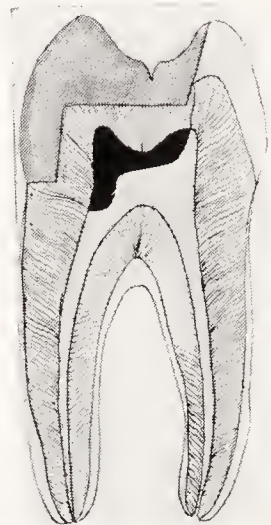


FIG. 57.—Diagram of the distribution of secondary dentin that would result from the preparation of a mesio-occlusal cavity and insertion of a filling in a lower molar. The secondary dentin is indicated by black shading.

Secondary dentin may be produced experimentally in animals by preparing cavities and placing fillings in them. It has been observed that secondary dentin formation in the teeth of young dogs begins without delay after a cavity has been drilled, and that after six weeks there is a considerable amount of it. Recent experiments of this kind, reported by Manley and by Gurley and Van Huysen, indicate that the amount of pulp reaction depends on the type of filling material and the depth of the cavity. Manley found the

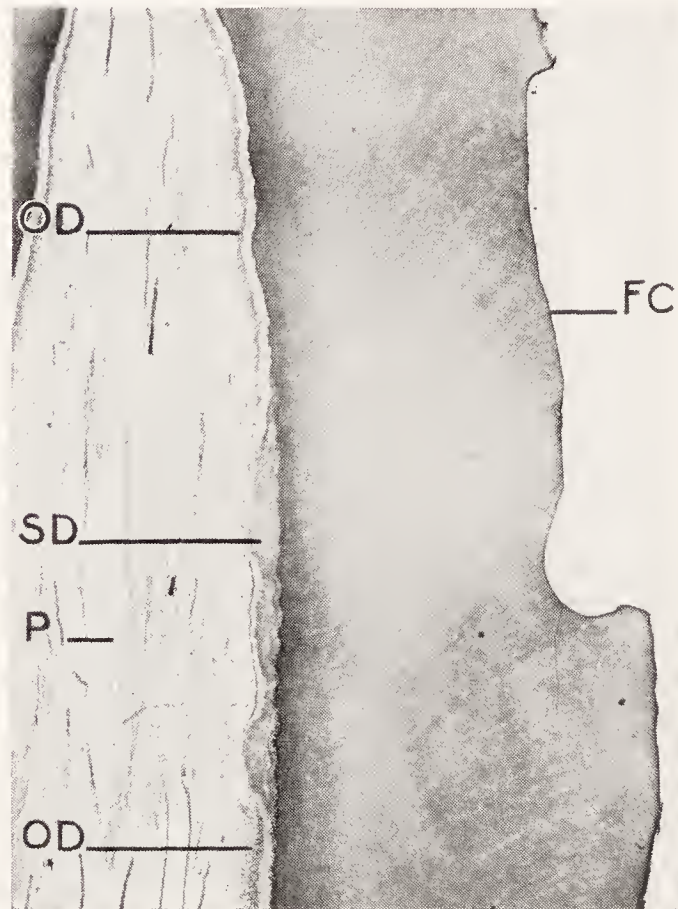


FIG. 58.—Secondary dentin under a shallow metal filling on the labial surface of an upper cuspid. *FC*, floor of cavity; *SD*, secondary dentin; *P*, pulp; *OD*, odontoblasts. Note the irregularity of the odontoblasts overlying the secondary dentin.

least reaction in the pulp when the cavity had been filled with zinc oxide and eugenol. More extensive secondary dentin formation and odontoblastic changes were observed under amalgam fillings. Silicate cements without a lining produced severe pulp changes and sometimes actual inflammation of the pulp. Gurley and Van Huysen confirmed Manley's observation that zinc oxide and eugenol produce the least pulp reaction of all filling materials; these seemed to have a palliative effect upon the exposed dentinal tubules. Other filling materials caused pulp reactions proportionate to the depth of the cavity, viz., the proximity of the filling to the pulp tissue.

A few specimens of human teeth that contained fillings over a period of several years will be used to illustrate the changes caused by such restorations. Figure 58 shows a labio-lingual section through

an upper cuspid that contained a shallow labial metal filling for one year. Secondary dentin corresponding to the extent of the cavity has been deposited on the labial wall of the pulp chamber. The odontoblasts overlying the secondary dentin are much smaller and more irregular than those on the surface of the primary dentin. The secondary dentin is located slightly rootward from the floor of the cavity; this is caused by the apical deviation of the dentinal tubules in their course from the cavity to the pulp chamber.

Figure 59 is an illustration of a mesio-distal section through a human lower molar, showing the outline of a mesio-occlusal amalgam filling that had been placed eight years before extraction. A mesial

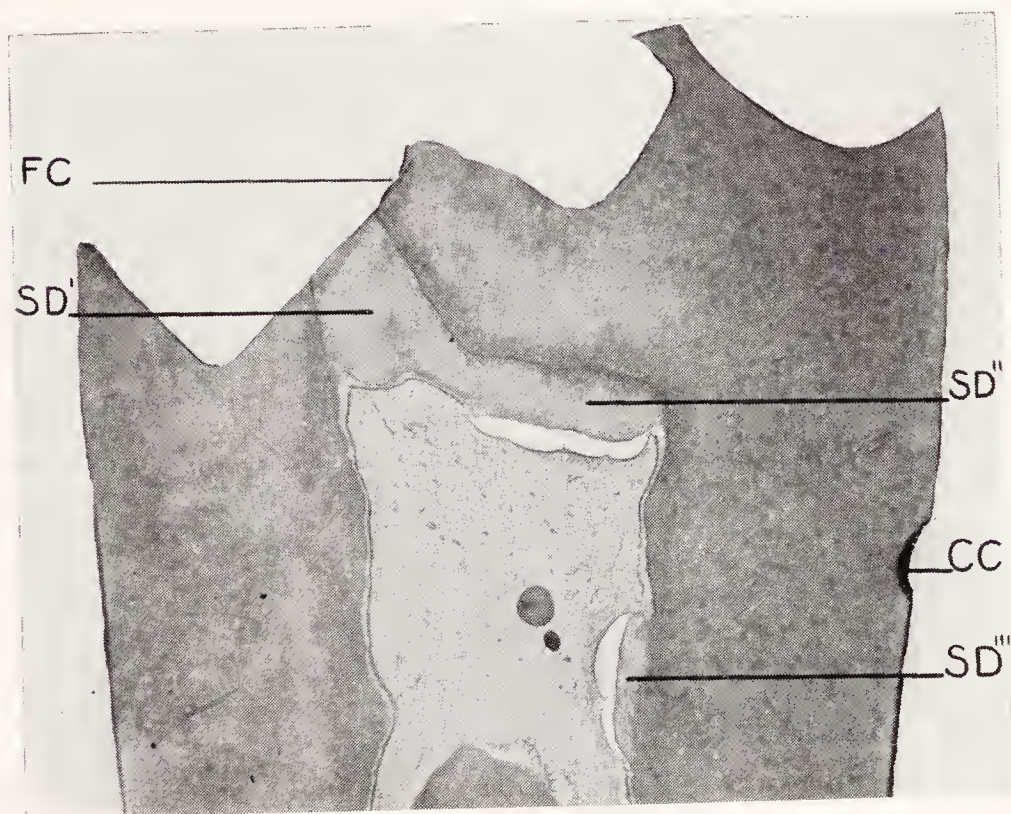


FIG. 59.—Secondary dentin under mesio-occlusal metal filling in lower molar. Filling inserted eight years before extraction. *FC*, floor of cavity; *SD'*, secondary dentin in mesial pulp horn; *SD''*, secondary dentin corresponding to the occlusal portion of the cavity; *CC*, cervical caries on the distal surface of the molar; *SD'''*, secondary dentin corresponding to this decayed area. (Coolidge, Illinois Dent. Jour.)

pulp horn has been completely obliterated by secondary dentin, which undoubtedly was already present at the time the cavity was prepared. There is also a deposit of secondary dentin on the roof of the pulp chamber corresponding in form and extent to the occlusal portion of the cavity. On the distal surface of the same tooth caries is beginning at the cemento-enamel junction, causing a circumscribed deposit of secondary dentin on the distal wall of the pulp chamber.

In Figure 60, also a mesio-distal section through a lower molar, the causal connection between cavity and secondary dentin is plainly indicated by the extent and arrangement of the latter. The

floor of the cavity almost reaches the outline of the original pulp chamber, and had it not been for the secondary dentin previously formed in this area, the pulp undoubtedly would have been exposed.

Tooth Fracture and Secondary Dentin.—A kick, blow, or other trauma may fracture the edge of an anterior tooth and expose the dentin. Unless the pulp is exposed or otherwise severely damaged, secondary dentin will be deposited in the pulp chamber; its distribution depends upon the location of the line of fracture. Usually, after a fracture, it is possible to observe the progress of secondary

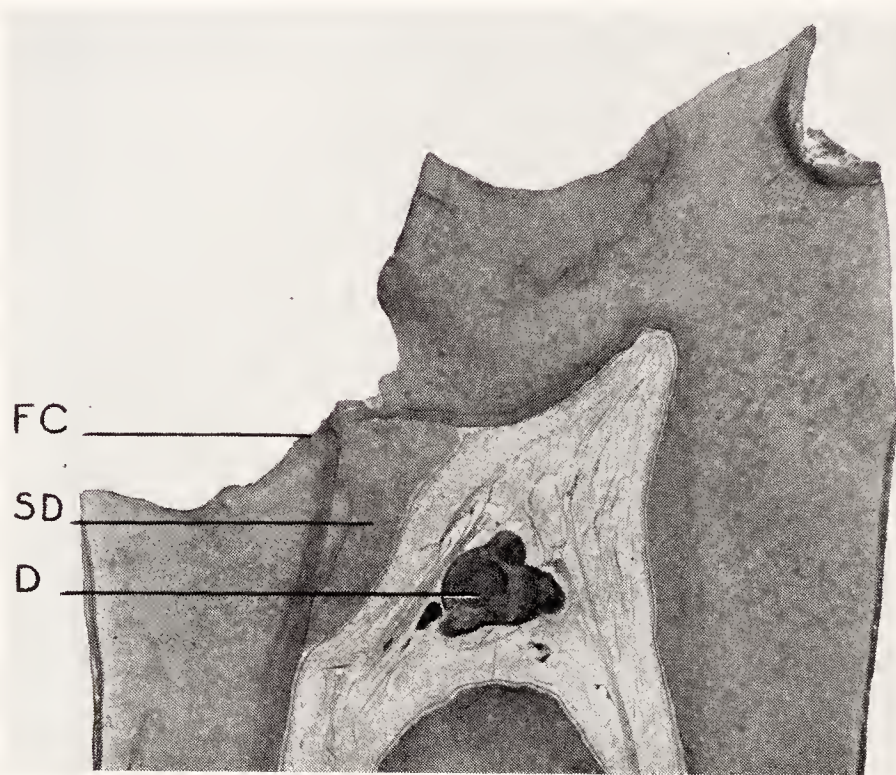


FIG. 60.—Secondary dentin under filling in lower molar. *FC*, floor of cavity; *SD*, secondary dentin in the mesio-occlusal corner of the pulp chamber; *D*, denticle. (Coolidge, Illinois Dent. Jour.)

dentin formation by means of radiographs: at first the pulp chamber shows no change; but after a few months to a year, it is considerably reduced in size, and after a few years it may be almost completely obliterated (see Chapter XVIII).

Pulp Atrophy and Secondary Dentin.—Most intact teeth of older individuals show dentin of irregular structure, at least in some areas. The walls of the root canals in practically all older teeth are formed by such dentin. External irritation may be partly responsible for this but cannot be the only cause, since irregular dentin occurs in completely embedded teeth and in teeth of ovarian cysts, neither of which are accessible to mechanical or other stimuli of the kind to which erupted teeth are subjected. The probable explanation for this irregular dentin is that the pulp tissue becomes atrophic and the odontoblasts degenerate with advancing age. The dentin

formed by a pulp of this kind is irregular and has the same structure as that laid down in response to external stimulation.

The large areas of irregular dentin that are frequently observed on the floor of the pulp chamber of intact molars also belong in this category. For unknown reasons the pulp tissue in this area undergoes atrophic changes and lays down dentin of irregular structure.

SCLEROSIS OF THE DENTIN.

The reaction of the tooth to external irritation is not confined to the formation of secondary dentin. The primary dentin, too, responds to injury or exposure of the dentinal tubules by becoming sclerotic. This reaction was originally described by W. D. Miller as transparency of the dentin, because dentin thus affected changes its optical qualities and in ground sections becomes more transparent to transmitted light than normal primary dentin. Beust reported a difference in the penetration of dyes into the dentin under different conditions. In young teeth the dentin stains easily from the pulp chamber outward to the dentino-enamel and dentino-cemental junctions. In adult teeth there are areas where the dye does not enter the tubules, and in old teeth most of the dentin may be impermeable to dye. Beust called the condition of impermeability of the dentin "sclerosis" (hardening) because it is an additional calcification of the dentin caused by obliteration of the dentinal tubules. The formerly patent tubules are filled with calcified material, and the dentin thus becomes impermeable.

Sclerosis of the dentin also occurs in response to external irritation. Under every carious cavity or abraded dentin surface is a sclerotic area leading to the pulp cavity (Figs. 61 and 62). This can be considered as a defensive reaction of the tooth, since the sclerosed dentin is impermeable and increases the resistance of the tooth to caries or to other external damaging agents. But external irritation is not the only cause of sclerosis, for sclerosed dentin is found in the teeth of individuals past middle age in areas that are not exposed or irritated in any way; it develops also in completely embedded teeth at about the same rate as in the erupted teeth of the same individual. Beust spoke of "tooth maturation" to describe this gradual age change in the dentin.

The problem of increased calcification with advanced age and as a result of dentin exposure has recently been studied by radiographic methods. Work along this line has been carried out by Van Huysen and his collaborators. They prepared thin ground slabs of uniform

thickness of intact, abraded, and carious teeth. When these slabs are photographed with roentgen-rays, the resulting picture indicates the degree to which the rays are absorbed in different areas. The enamel shows the greatest absorption; it has the highest content of inorganic material and absorbs nearly twice as much radiation as dentin. In normal, unchanged dentin the absorption is almost uniform; however, dentin located under abraded surfaces or under decay has a greater density than normal dentin. In some areas under carious cavities the roentgen-ray absorption of the dentin increased



FIG. 61. — Cuspid showing opaque sclerosis under abraded occlusal surface. Stained section, photographed by transmitted light. (Beust, Jour. Dent. Res.)



FIG. 62. — Same specimen as that shown in Fig. 61, but photographed by reflected light. The opaque sclerosed areas appear white. (Beust, Jour. Dent. Res.)

from 10 to 25 per cent as compared to normal dentin. These findings by Van Huysen indicate that the formerly only assumed greater calcification of sclerosed dentin actually exists.

A different approach to the problem of dentin calcification is that reported by Hodge and McKay who investigated the hardness of tooth tissues by a scratch method. A fine diamond point is passed with uniform pressure across the surface to be tested; the diamond cuts a fine groove, and the depth and width of this groove are inversely proportionate to the hardness of the material tested. The findings obtained by this method suggest the presence of a zone of considerably increased hardness adjacent to the dentin softened by decay.

Sclerosis of the dentin is of great practical importance. It is a contributory cause for the decrease in the sensitiveness and perme-

ability of human teeth with advancing age. Together with secondary dentin formation, sclerosis aids in sealing the dentin under abrasion, erosion, and caries, and thus aids in preventing pulp irritation and pulp infection.

REGRESSIVE CHANGES IN THE PULP TISSUE.

The term "regressive changes" is used for certain conditions in the pulp tissue that are usually associated with the formation of secondary dentin and, like the latter, are on the borderline between normal and pathological. Such changes are invariably found in all teeth of older persons. Even intact teeth of young individuals, as a rule, are not altogether free of them; at least in some areas, they show incipient regressive changes, atrophy of the pulp tissue or degeneration of the odontoblasts.

Regressive changes in the pulp do not cause any clinical discomfort and can be seen only under the microscope; clinically the tooth may be healthy and intact. This is an important point because here, as in many problems, the dentist's clinical conception is not in accord with the pathologist's microscopic diagnosis. The dentist considers a tooth normal and healthy if the crown is intact, if the pulp reacts to vitality tests, and if there is no evidence of pathological changes in the supporting structures. The pathologist, in examining the pulp of the same tooth under the microscope, may find degenerative changes, vacuolization of the odontoblasts, atrophy and calcification of the pulp tissue, and may not consider it normal. The dentist's diagnosis, however, is the correct one.

The first symptom of the beginning of degenerative changes in the pulp is the presence of fat in the form of fine droplets in the pulp tissue. These deposits may be found in the odontoblasts as well as in the nuclei of the pulp cells and in the walls of the pulp capillaries. Further degenerative changes in the pulp are vacuolization of the odontoblastic layer and atrophy of the pulp tissue. Both processes are characterized by a decrease in the number and size of the pulp cells and their replacement by fibers (fibrosis of the pulp).

Figure 63 shows the process of vacuolization of the odontoblasts. In the center of the field the odontoblasts are still visible as a continuous row of elongated cells; above and below, however, they are pushed apart by an intercellular accumulation of fluid. The pressure of this fluid has dislodged some of the odontoblasts from the dentin surface.

Incipient atrophy of the pulp tissue is illustrated in Figure 64. There are fewer cells than in a normal pulp, and there are small, round, fluid-containing areas between them. As a result of these

changes, the pulp tissue has the appearance of a net, and, therefore, this condition is called reticular atrophy. As reticular atrophy advances, the spaces or vacuoles become larger, and the number of

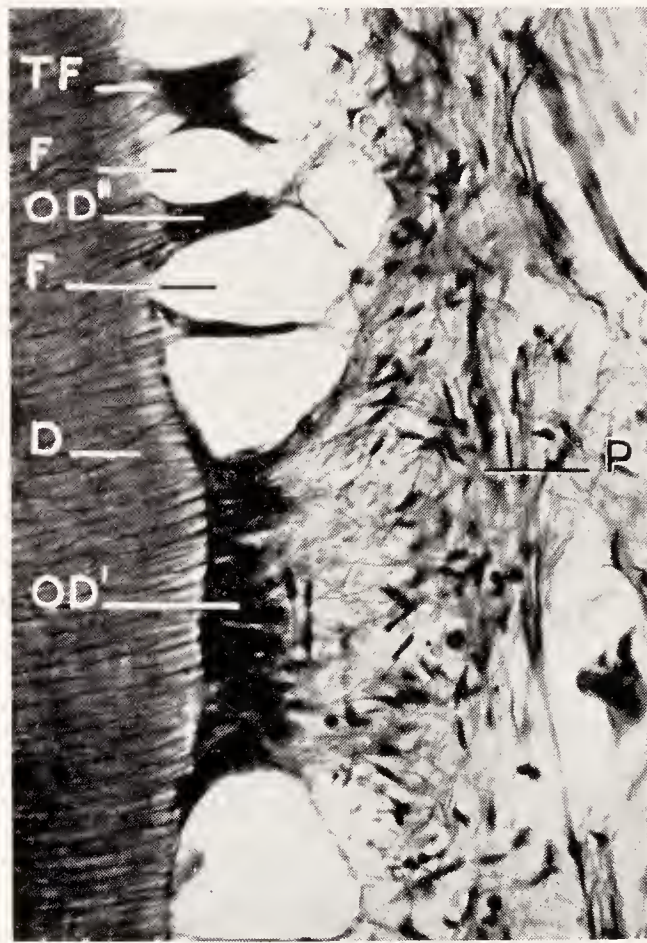


FIG. 63.—Vacuolization of the odontoblastic layer. *OD'*, normal odontoblasts; *F*, accumulation of fluid between the odontoblasts; *OD''*, groups of odontoblasts between the vacuoles. The connection between the odontoblasts and the ends of Tomes' fibers, *TF*, can be plainly see; *D*, dentin; *P*, pulp. (Courtesy of W. Willman.)

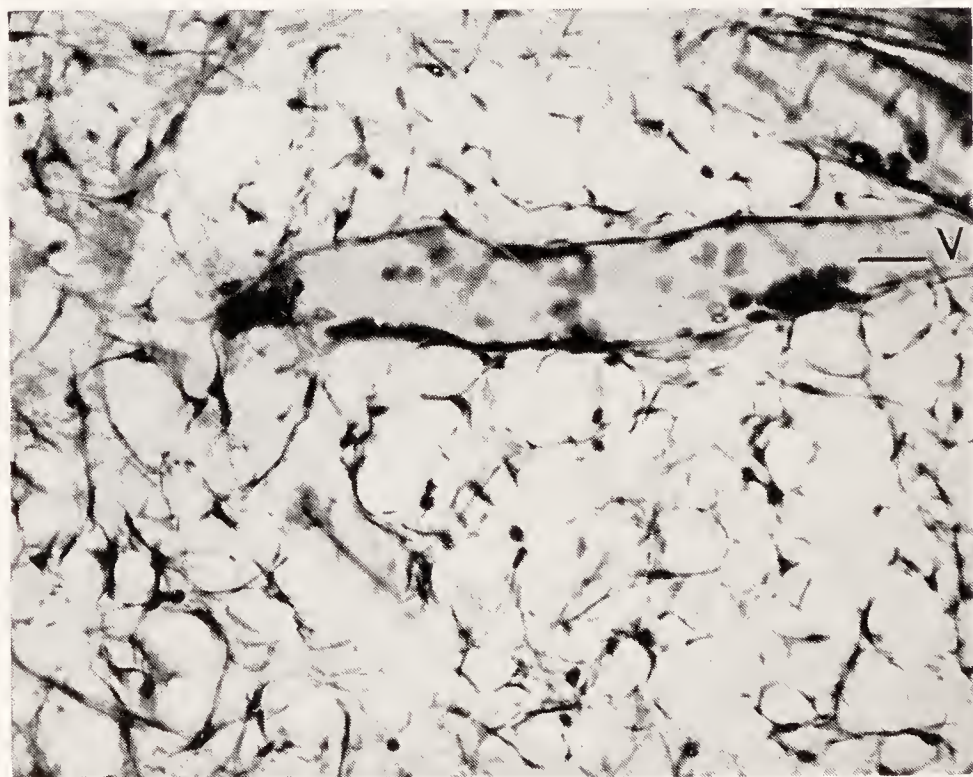


FIG. 64.—The beginning of reticular atrophy of the pulp. Reduction in the number of pulp-cells. The intercellular accumulation of fluid gives the pulp a net-like appearance. *V*, blood-vessel. (Courtesy of W. Willman.)

cells is reduced (Fig. 65). In extreme cases, few cellular elements are left; the pulp tissue consists of irregular, coarse strands of fibrous tissue. The number of blood-vessels and nerves is greatly reduced,

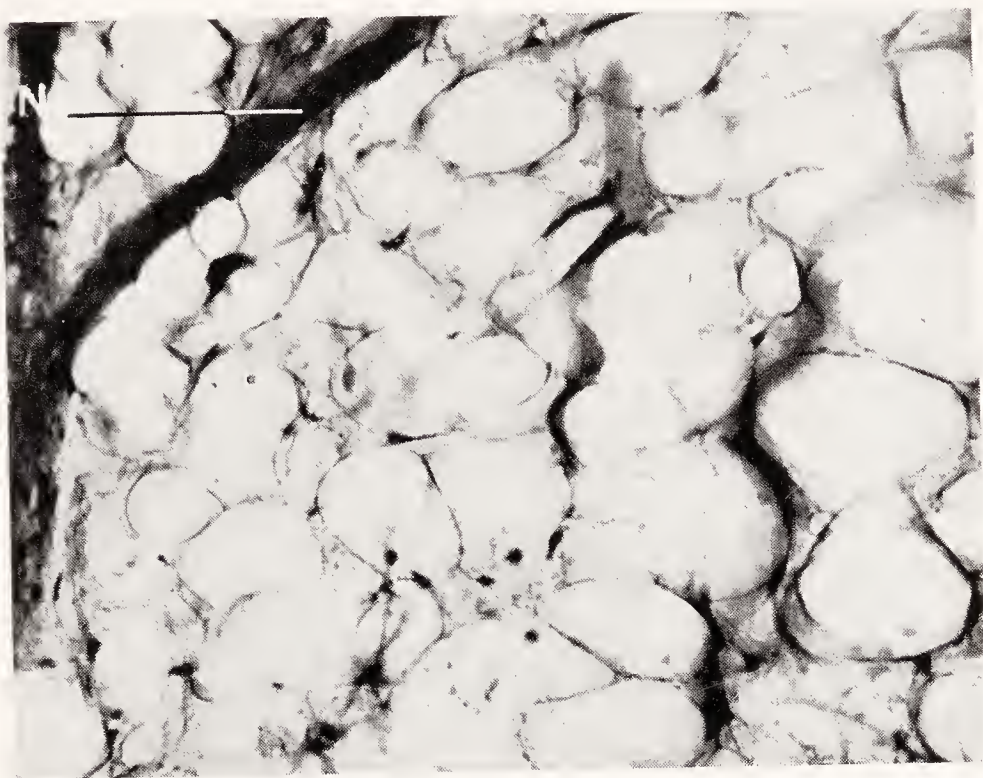


FIG. 65.—Reticular atrophy of the pulp. Formation of spaces in the fibrous pulp tissue. Greatly reduced number of pulp cells. *N*, nerve. (Courtesy of W. Willman.)

which accounts for the decreased sensitiveness of such pulps. The odontoblasts have completely disappeared (Fig. 66). Any dentin

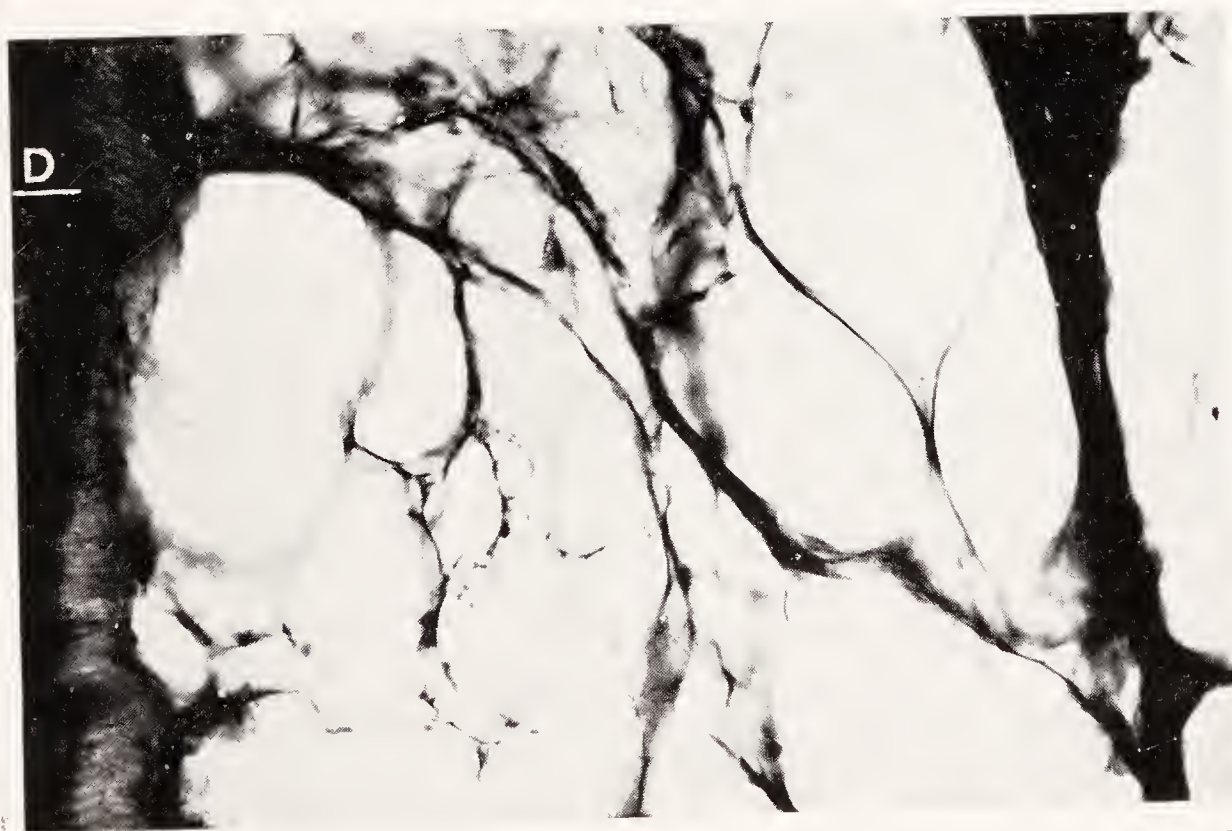


FIG. 66.—Advanced reticular atrophy of the pulp. Only a few coarse strands of fibrous tissue are left. Very few cellular elements are present. *D*, dentin. (Courtesy of W. Willman.)

formed by such a pulp is very irregular in structure, with only a few atypical dentinal tubules.

Closely related to pulp atrophy is the occurrence of so-called pulp cysts in the pulps of intact teeth. They are fluid-containing spaces of varying size surrounded by fibrous pulp tissue, but they are not true cysts since they lack an epithelial lining. Probably they are the result of localized pulp atrophy and shrinkage of the pulp tissue (Schneider).

CALCIFICATION OF THE PULP.

Abnormal calcification occurs very frequently in human pulp tissue. Pulp calcifications can be classified according to size, structure, and locality. Relatively large, well-outlined bodies of hard substance found in the pulp chamber are called denticles, pulp stones, or pulp nodules. In addition, fine diffuse calcifications are frequently observed: they are spoken of as fibrillar calcifications or calcific degeneration of the pulp tissue.

Denticles (Pulp Stones, Pulp Nodules).—According to their microscopic structure denticles are classified as true and false. True denticles are those that consist of irregular dentin. False denticles are degenerative calcifications of the pulp tissue; under the microscope, they usually show a concentric, lamellated structure as the result of the deposition of consecutive layers of calcium salts around a central nucleus.

According to their relation to the walls of the pulp chamber, denticles are classified as free, adherent, and interstitial. Free denticles lie free in the soft tissue of the pulp without connection with the walls. Adherent denticles are attached to the wall of the pulp chamber. Interstitial denticles are embedded in dentin. This latter type develops from free or adherent denticles by continued dentin formation, which finally entirely surrounds the denticle.

Large denticles can be diagnosed by means of the radiograph, which may reveal whether they are adherent to the wall of the pulp chamber or lying free in the pulp tissue. Their size varies from hardly visible, dust-like granules to 2 or 3 mm., filling almost the entire pulp chamber. If the pulp chamber is opened, denticles can sometimes be removed with a spoon excavator (free denticles); other times they are solidly united with the walls of the pulp chamber (adherent denticles) and then may cause difficulties if treatment of the root canals is intended. Large pulp stones in the pulp chamber can make the root canals almost inaccessible; those adhering to the

walls of the root canals sometimes form an insurpassable obstacle to broaches or reamers.

True Denticles.—Denticles with the histological structure of dentin are the result of a localized excess formation of dentin. They usually develop from the wall or from the bottom of the pulp chamber because of excessive dentin-forming activity in a circumscribed area, and are closely related to secondary dentin in structure. Figure 67

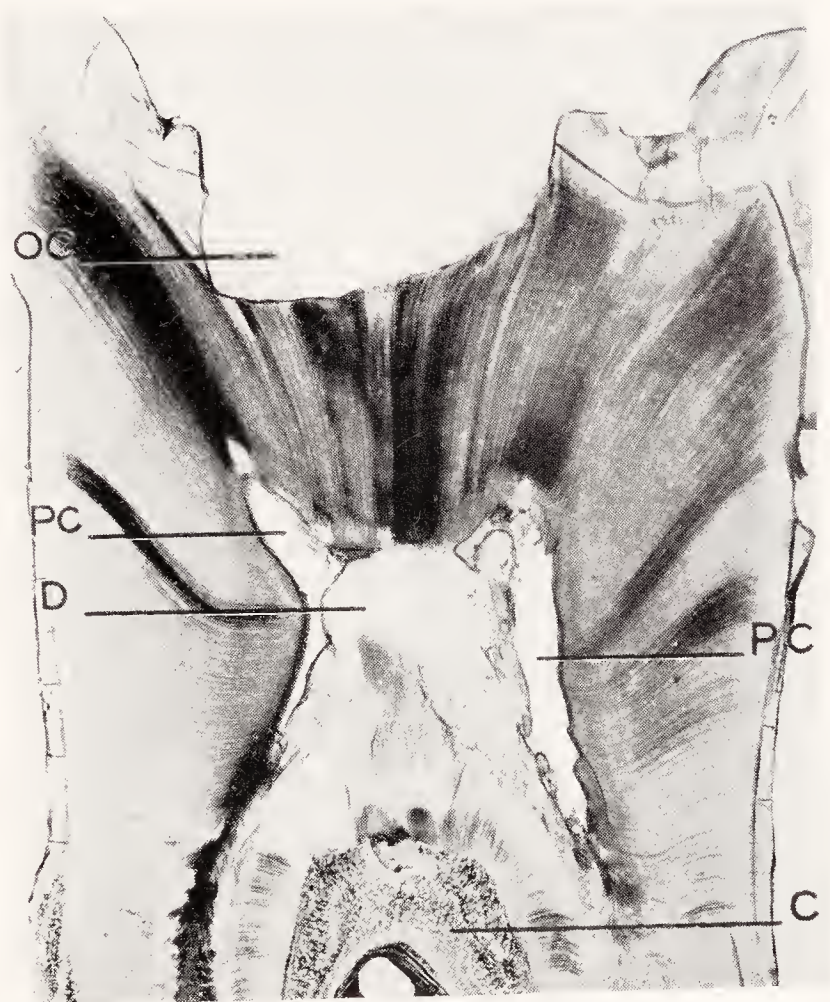


FIG. 67.—Large true denticle in the pulp chamber of lower molar. Ground section. The denticle is attached to the floor and to the roof of the pulp chamber, thus dividing the pulp chamber into a mesial and a distal portion. *OC*, occlusal cavity; *D*, denticle; *PC*, pulp chamber; *C*, cementum in bifurcation.

shows a photomicrograph of a ground section through a lower molar. A large true denticle developed from the floor of the pulp chamber and grew toward the roof until it came in contact with the latter, thereby dividing the pulp chamber into two small sections. The denticle consists of secondary dentin with scanty, irregular dentinal tubules (Fig. 68).

Sometimes true denticles lie free in the pulp chamber, entirely surrounded by pulp tissue. However, such free true denticles are rare; histologically most free denticles are false denticles. For the formation of a free true denticle with dentinal tubules, it is necessary for odontoblasts to develop within the pulp tissue. Apparently pulp cells may occasionally be transformed into onto-

blasts and take part in the formation of dentin in pulp stones. The presence of epithelial cells in the pulp may have some connection with this type of dentin formation.

Figure 69 shows a free true denticle. The inner part of it consists of calcified pulp fibers, the outer part of irregular dentin; the surface



FIG. 68.—High magnification of the denticle in Fig. 67. It consists of secondary dentin with irregular dentinal tubules, *DT*.

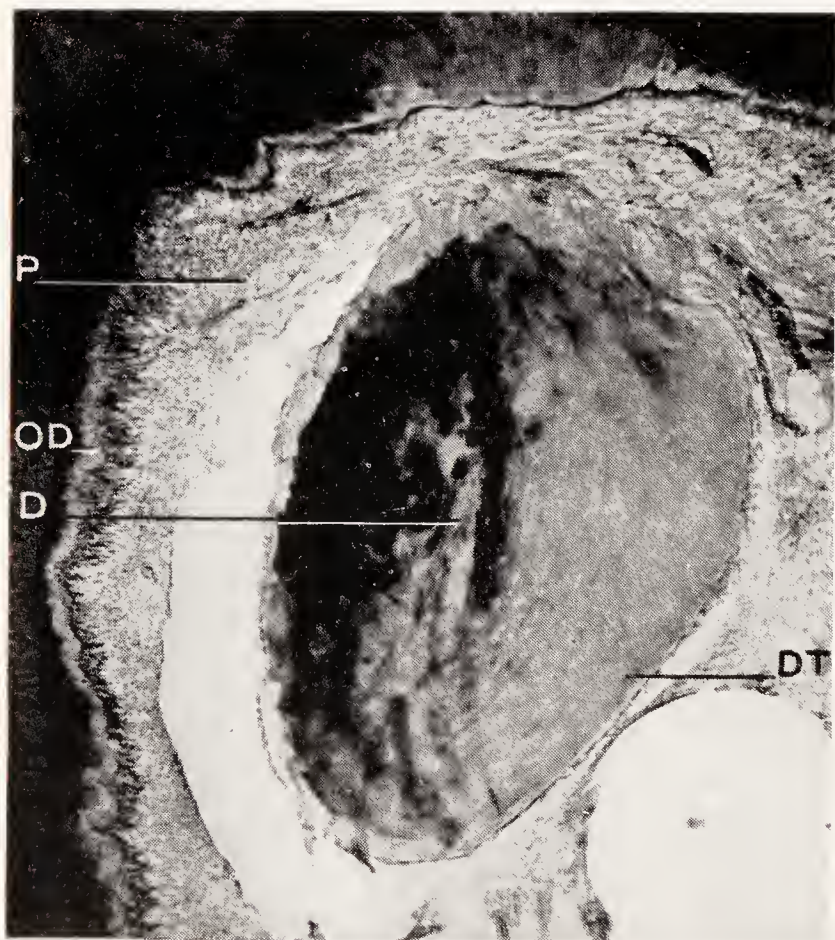


FIG. 69.—Free true denticle. *D*, denticle; *DT*, dentinal tubules in the denticle; *P*, pulp tissue; *OD*, odontoblasts.

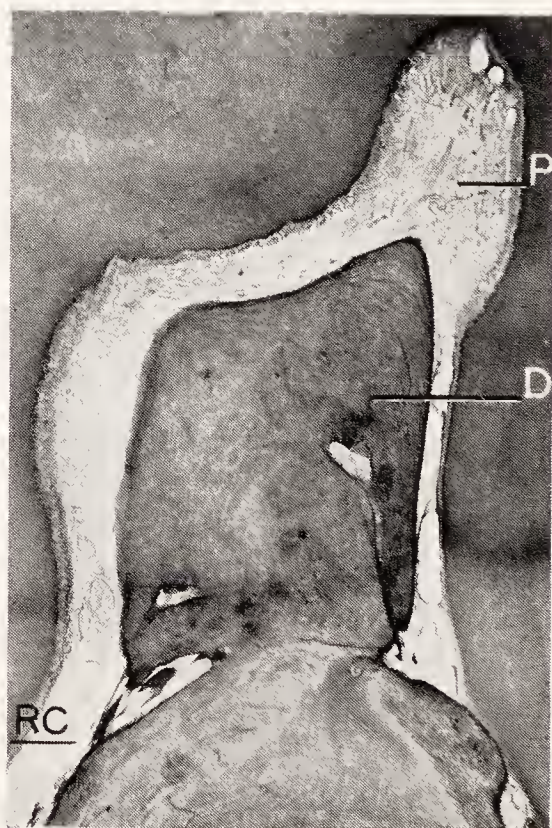


FIG. 70.—Large denticle attached to the floor of the pulp chamber of an upper molar. The pulp chamber is reduced to a narrow slit between denticle and dentin wall. *D*, denticle; *P*, pulp; *RC*, root canal.

is covered with a layer of predentin and is surrounded by odontoblasts. Evidently the calcification of the pulp fibers took place first, these calcified masses forming the nucleus around which irregular dentin was later deposited.

False Denticles.—Most of the pulp stones found in human teeth are histologically false denticles. They originate from deposits of calcium salts in the pulp tissue, probably following minor circulatory disturbances in the pulp vessels. They vary greatly in size; sometimes they fill almost the entire pulp chamber. Figure 70 illustrates a large denticle that is attached with a broad base to the

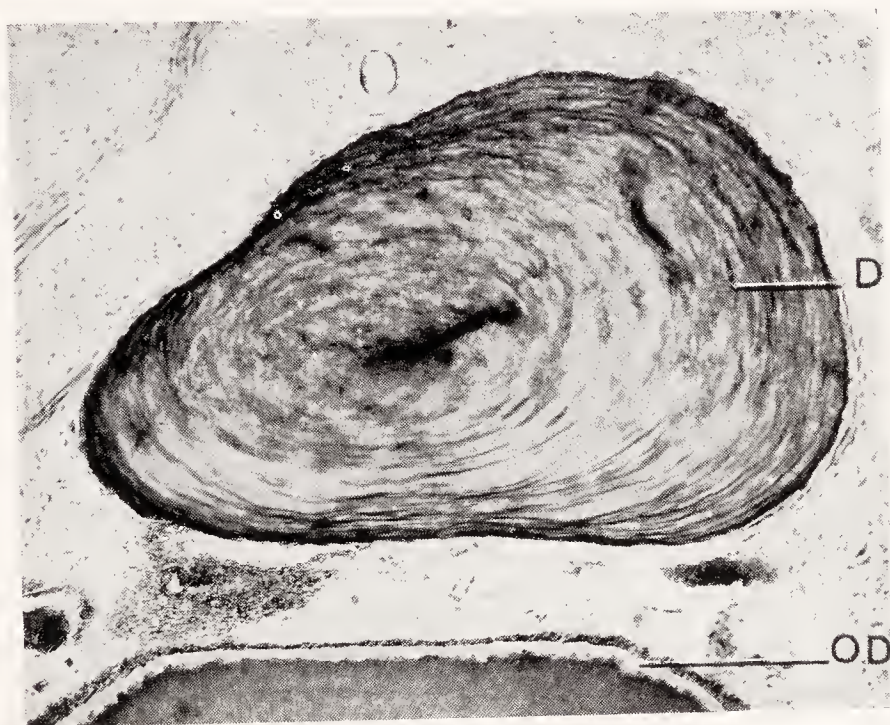


FIG. 71.—High magnification of a small free denticle of concentric lamellated structure (false denticle). *D*, denticle; *OD*, odontoblasts and dentinoid on the floor of the pulp chamber. Notice the strand of calcified fibrous tissue in the center of the denticle. This calcified tissue forms the nucleus around which the denticle is built in concentric layers.

floor of the pulp chamber. The pulp tissue is confined to a narrow space around the denticle, between the latter and the wall of the pulp chamber.

False denticles are formed by the deposition of consecutive layers of calcium salts around a central nucleus, which usually consists of some degenerated, hyalinized pulp tissue (Fig. 71). As these round denticles grow, they approach each other and may finally become fused. If they are close to the wall of the pulp chamber, they gradually become adherent by a solid union with the dentin (Fig. 72).

Two small, false denticles in the root canal of a lower molar are illustrated in Figures 73 and 74. In Figure 73 the denticle is attached to the wall of the root canal. Evidently it was originally free in the pulp tissue; later on, when the lumen of the canal was gradually

reduced by continued formation of dentin, it became adherent. As the deposition of dentin continues, such a denticle may finally be entirely surrounded by dentin, and it then becomes an interstitial denticle. This condition is shown in Figure 74: a round denticle of concentric, lamellated structure is completely embedded in the wall of the root canal. These denticles are usually small and of little practical importance.

Pulp Calcifications (Calcific Degeneration of the Pulp Tissue).—Fine, fibrillar calcifications are abundant in some pulps, especially

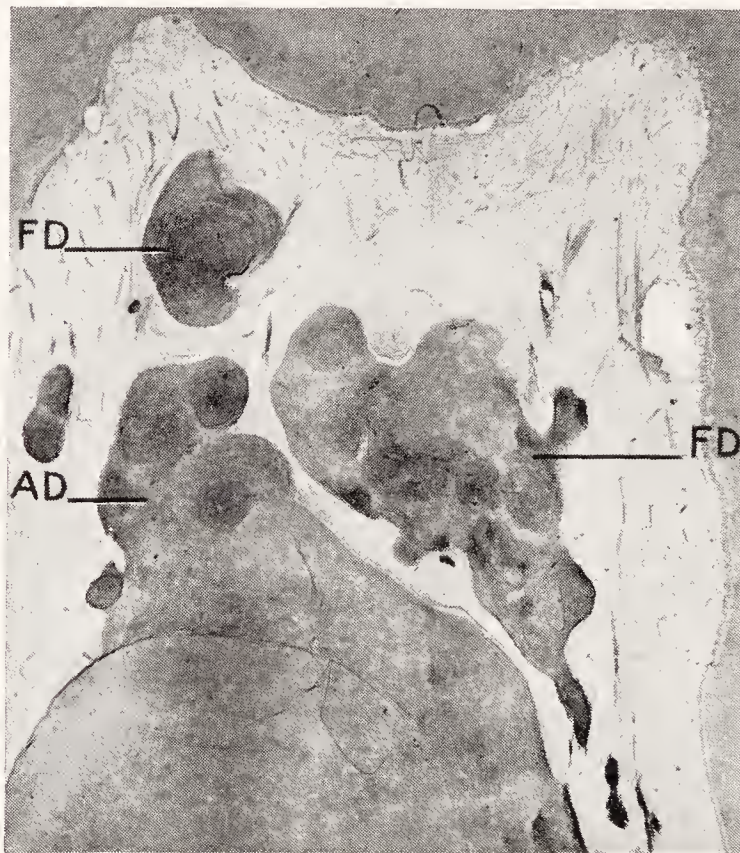


FIG. 72.—Several free and adherent denticles in lower molar. The larger denticles are the result of a coalescence of several smaller ones of concentric lamellated structure (false denticles). *FD*, free denticles; *AD*, adherent denticle on the floor of the pulp chamber.

in those of older teeth (Fig. 75). Sometimes such calcifications begin in the wall of a blood-vessel or in the perineural connective tissue of a larger nerve, and then follow the course of the strands of vessels and nerves in the pulp. By the fusion of such perivascular or fibrillar calcifications, long, thin denticles are formed, the origin of which can easily be recognized under the microscope (Fig. 76).

In the walls of the pulp vessels of adults and old persons, diffuse deposits of lime salts are observed (Euler). These may be the only calcifications of this kind in the body, or they may be an expression of a generalized arteriosclerosis.

Incidence of Pulp Calcifications.—The frequency with which pulp stones and other pulp calcifications occur in human teeth has been studied by Willman and by Hill. Both arrived at approxi-

mately the same conclusions. Willman found pulp calcifications in 143 out of 164 teeth, or in 87.2 per cent. Hill reported pulp calcifications in 66 per cent of all teeth from individuals between ten and twenty years of age, and 90 per cent between the ages of fifty and seventy years. Willman pointed out that only about 15 per cent of all pulp calcifications are large enough to be seen in radiographs. This is important because it shows that only one out of every six pulp stones can be diagnosed clinically, or, in other words, for each pulp

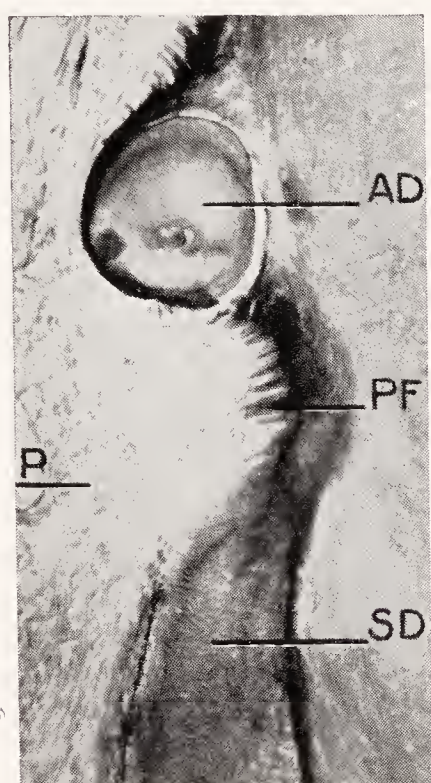


FIG. 73.—Small adherent denticle in the root canal of a molar. *P*, pulp; *AD*, adherent denticle; *SD*, secondary dentin; *PF*, calcifying pulp fibers.

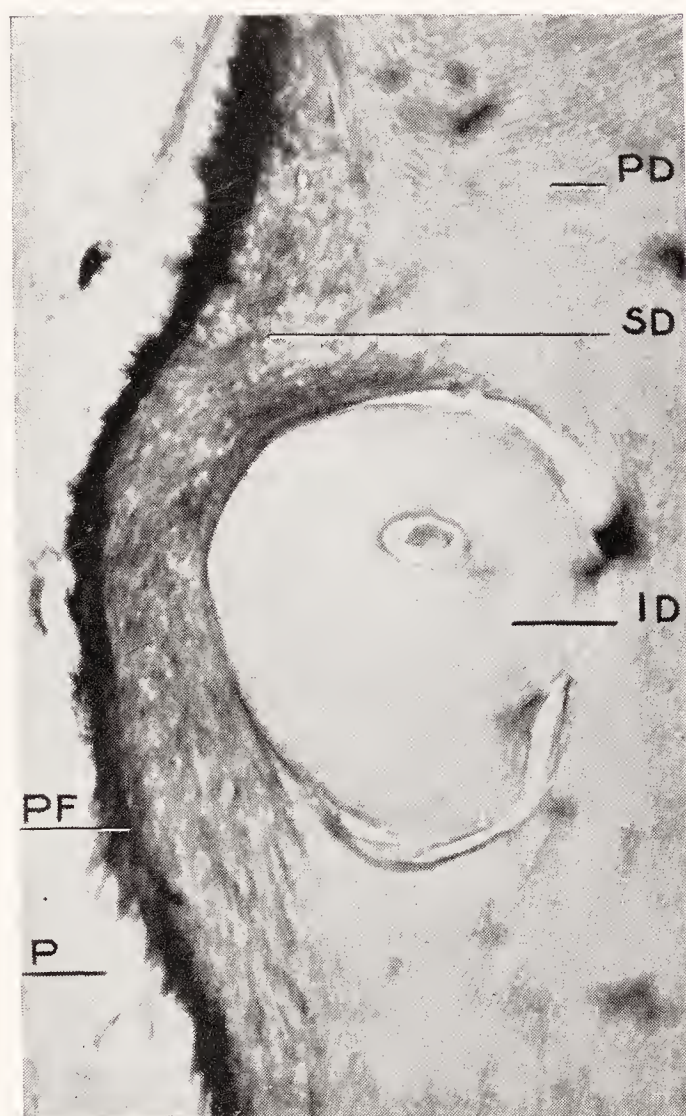


FIG. 74.—Interstitial denticle in the wall of the root canal of a molar. *P*, pulp; *ID*, interstitial denticle; *PD*, primary dentin; *SD*, secondary dentin; *PF*, calcifying pulp fibers. The denticle shows a concentric laminated structure around a central nucleus.

stone seen in a radiograph at least six others are present, but they are so small that they are visible only microscopically. The higher incidence of pulp stones in older teeth may be explained by their gradual increase in size: developing from a small center of calcification early in life, a pulp stone grows until it reaches a size at which it is easily visible both radiographically.

Etiology of Denticles and Pulp Calcifications.—The formation of denticles is often associated with the presence of an irritant of long standing, such as caries or deep fillings. They occur more often in old people than in young ones. Denticles have been found, how-

ever, in very young teeth and even in tooth germs before eruption; therefore, outer irritations cannot be the only cause for their formation. Because of the absence of any collateral circulation, minor circulatory disturbances frequently occur in the pulp. As a result, the connective tissue of the pulp often shows small areas of hyalinization, which later become calcified and form the nuclei for the formation of denticles.

The presence of denticles is sometimes held responsible for pulpitis pain or even trifacial neuralgia. In some histological specimens,

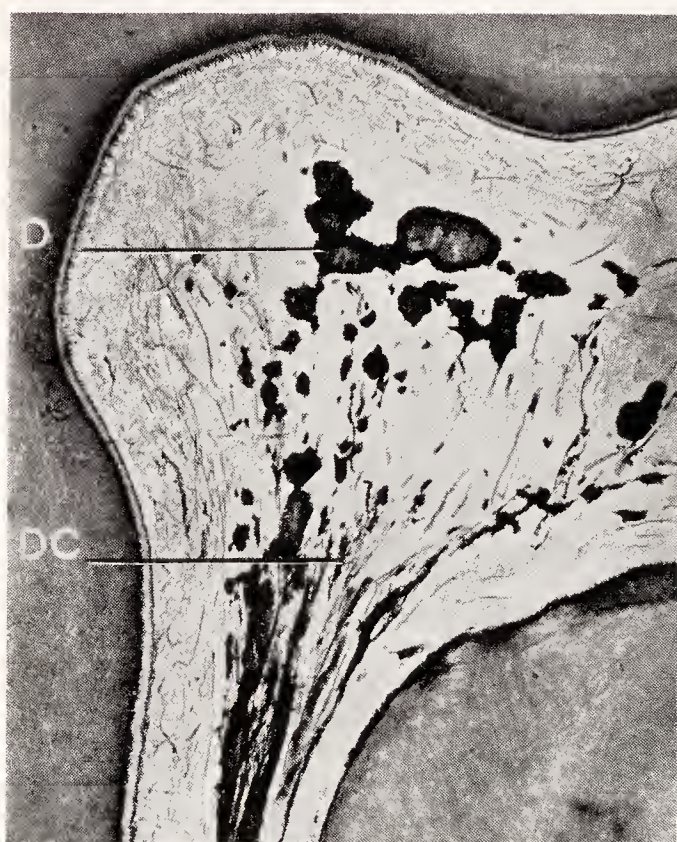


FIG. 75



FIG. 76

FIG. 75.—Diffuse calcification (*DC*) of the pulp tissue in an intact lower molar of an adult. *D*, denticles resulting from fusion of minute calcified areas.

FIG. 76.—Fibrillar calcification of the pulp tissue in a root canal. *P*, pulp tissue; *OD*, odontoblasts and dentinoid; *PF*, calcified pulp fibers; *D*, denticle formed by coalescence of calcified pulp fibers; *VO*, vacuolization of the odontoblastic layer.

denticles are found in close proximity to pulpal nerves, and it is not impossible that these nerves may become irritated by the growing denticle. However, in clinical practice it is necessary to study and analyze all circumstances most carefully and to rule out all other possible causes for neuralgic pain before a denticle can be looked upon as the etiological factor.

Pulp stones never cause pulp inflammation, and there is no justification for considering them a source of dental infection. Bacteria have been obtained by cultural methods from pulp stones in intact teeth. These bacteria either originate from contamination during extraction of the tooth (Kanner), or they belong in the same cate-

gory as bacteria that are occasionally found in the pulps of healthy intact teeth and that have no pathogenic significance. The mere presence of bacteria in the pulps of intact teeth, with or without pulp stones, must not be interpreted as infection unless these pulps show evidence of an inflammatory reaction (Tunnick and Hammond).

METAPLASIA OF THE PULP.

Metaplasia is the transformation of one tissue into another. Metaplasia of the pulp means a change in the structure and function of the pulp tissue. Normally the pulp forms dentin, whereas the

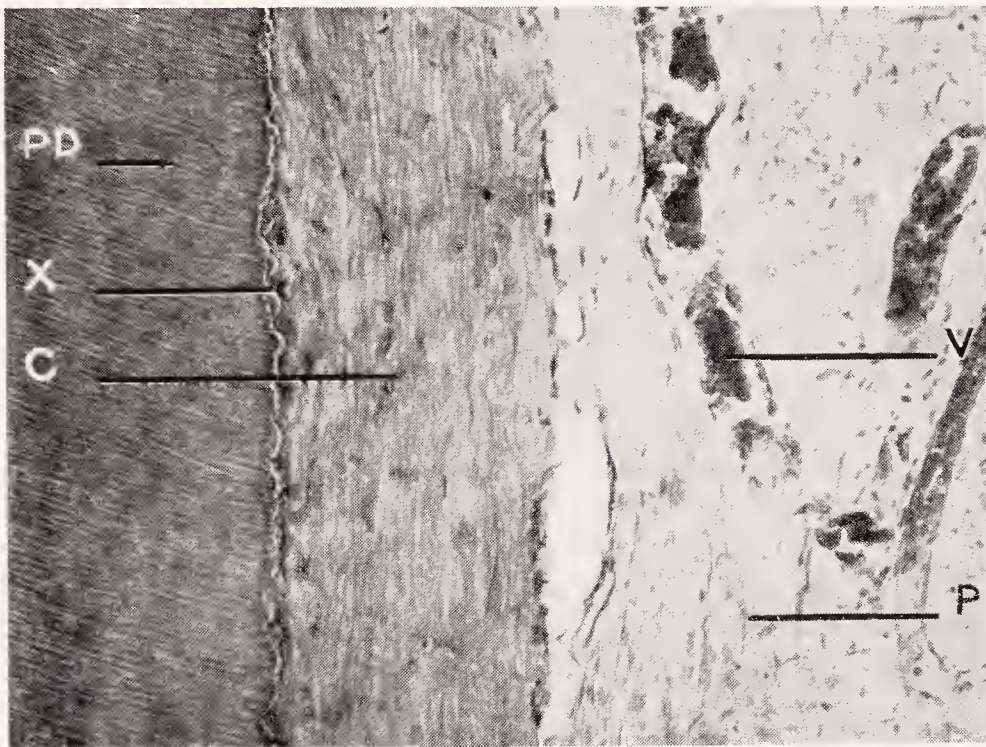


FIG. 77.—Metaplasia of the pulp. Intact upper third molar. Deposition of cementum on the wall of the pulp chamber. *PD*, primary dentin; *X*, borderline between dentin and cementum; *C*, cell-containing cementum; *P*, pulp tissue; *V*, blood-vessels of the pulp.

connective tissue of the jaw forms bone or cementum. In case of metaplasia, the pulp changes its function and forms cementum or bone instead of dentin; then bone or bone-like cementum is found in the pulp chamber.

A case of metaplasia of the pulp is illustrated in Figure 77. In examining sections through an intact upper third molar of an adult, the pulp chamber was found completely lined by a layer of cell-containing cementum that was separated by a definite line from the dentin. Nothing is known about the history of this tooth, and nothing in the specimen indicated any external disturbance; for some unknown reason the pulp changed its function from the formation of dentin to the formation of a hard substance having the characteristics of bone.

True metaplasia of the pulp is a rare condition; it must not be confused with the frequent observation of cementum in the apical portion of root canals following chronic pulpitis, root canal treatment, or pulp injuries. These deposits of cementum are the result of a proliferation and ingrowth of periodontal connective tissue through the apical foramen into the root canal, and of substitution of the pulp tissue by connective tissue.

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CHAPTER V.

DENTAL CARIES.

CLINICAL CHARACTERISTICS OF DENTAL CARIES.

THE fundamental facts concerning dental caries as compiled by Bunting are given in the following paragraphs:

“1. Dental caries is a destruction of the hard substances of the tooth by a process, the initial stage of which is a decalcification by acids.

“2. The acids active in caries are not generally distributed in the saliva, but are localized and concentrated on certain areas of the tooth surfaces.

“3. Carious lesions occur most frequently in the pits and fissures of the occlusal surfaces and on certain areas of the approximal, buccal, and lingual surfaces of the teeth, at which locations there is opportunity for stagnation and the retention of foreign matter. They do not occur on smooth enamel surfaces which are frequently cleansed.

“4. All initial lesions of caries contain acid-forming bacteria capable of producing and living in acids of sufficient potential to decalcify the enamel.

“5. The hardness or softness of the teeth may affect the rate of progress and extent of caries but do not alone determine its occurrence. Caries, as a rule, runs a more rapid and extensive course in poorly formed teeth than in the hard and well-formed varieties, but instances commonly occur in which the poorest formed teeth are wholly free from the disease.

“6. Malhygiene of the mouth frequently favors the inception of dental caries and increases its activity, but alone does not determine its occurrence. Mouths that are habitually unclean are often wholly free from caries and, conversely, mouths that are scrupulously clean may be seriously affected by the disease.

“7. The process of dental caries is related to and often determined by certain constitutional states and conditions of bodily health. The nature of these general influences and the manner in which they affect the course of this dental disease are not clearly understood at this time. The following bodily conditions are perhaps best known as systemic factors which either favor or oppose dental caries:

“(a) *Heredity*.—There are strong evidences that the tendency toward dental caries or an immunity to the disease may be transmitted from parent to child, according to the laws of familial inheritance.

“(b) *Susceptibility* to dental caries is clearly influenced by age. Incidence of the disease is known to be highest during the ages of seven to twenty years. After twenty years the tendency to caries is markedly decreased.

“(c) *Health*.—It is frequently noted that severe onsets of dental caries follow attacks of general disease and disturbance of bodily health. During pregnancy caries may be unusually active. Children who are undernourished or are suffering from a general debility are specially prone to dental caries.

“(d) *Racial Influences*.—Dental caries is more prevalent in certain races than in others. The natives of Africa, South America and the South Sea Islands, the Eskimo and many other primitive peoples are notably free from the disease, while those who live in the more civilized communities are extremely susceptible to it. There are evidences that the disease often increases in prevalence as the people advance in the scale of civilization. It is also observed that when individuals migrate from a caries-free nation to a country in which it is prevalent, they and their progeny may later develop dental disease. There is no indication that dental caries is an endemic affection or that it is induced by any particular climatic conditions. The most constant and important variable between immune and susceptible races appears to be that of diet.”

EXPERIMENTAL RESEARCH IN DENTAL CARIES.

Influence of Dietary Conditions Upon Dental Caries in Children.

—Investigations dealing with the frequency and progress of dental decay in man can be carried on only where large groups of children live in closed institutions (schools, orphanages), so that it is possible to control the food intake of every child over a period of several years. During this time the children live under a definite dietary régime, which can be varied in order to produce the desired experimental conditions. The amount of tooth decay present in the teeth of these children is carefully checked and tabulated and compared with figures obtained from the examination of control children who live under different dietary conditions.

At the present time such studies on large groups of children are being made in several parts of this country. Although the different

investigators are not entirely in accord as to exactly what are the effective factors in the control of dental caries, they all agree that the teeth of a child on an adequate and well-balanced diet are less subject to decay than those of a child who is fed on an average or poor diet. The results obtained by these investigators are highly encouraging, for it seems actually possible to reduce the incidence and the progress of dental caries by an adequate dietary régime.

As an example of an experiment of this kind, a few of the actual findings of the Michigan group of investigators (R. W. Bunting, Philip Jay, and their associates) at a county orphanage are described here. In accompanying Dr. Bunting on his semi-annual dental check-up in the institution, the author had an opportunity to examine the mouths of more than 60 children; the incidence and extent of dental caries were compared with the accurate charts of the dental conditions of these children one year before. The healthy state of the mouths and the small amount of caries were remarkable. In many instances cavities that had been recorded a year before had not changed in size, and the bottom of them appeared hard when examined with an explorer. A careful analysis of the diet of these children revealed certain inadequacies from a nutritional point of view. The diet was below the minimum standard requirements in calcium, phosphorus, protein, and some vitamins. Sweets and cookies were served only on rare occasions; no candy was allowed at any time. Only a minimum amount of sugar was used in cooking, and no sugar was available at the table.

The following observations were made in this institution over a one-year period: Of 159 children 119, or 75 per cent, showed no evidence of active caries. In this group there were no new cavities, and unfilled cavities present the year before had not become larger. Active, new caries appeared in only 7 children in the form of one to three small cavities or extensions of previous carious lesions. In the remaining 33 children caries was either questionable or very slight. This percentage of active caries is far below that which every practitioner or pediatrician is able to observe among average American school children. Therefore, it seems that the favorable conditions found under certain food régimes actually can be attributed to the dietary factor.

Control experiments with a nutritionally adequate, high-carbohydrate diet showed a high incidence of caries. Bunting pointed out that the caries incidence in children on an inadequate low sugar diet is lower than in those on an adequate high sugar diet. He concluded that in a great majority of cases dental caries can be

definitely arrested by the adoption of a fairly adequate low sugar diet.

Boyd and Drain at the University of Iowa observed during routine mouth examinations in a children's clinic that a group of 28 children showed arrest of dental caries. These children had open, untreated cavities that showed no tendency to increase in size; the walls of the cavities had become hard and dark. Mouth hygiene apparently had no relation to this change since some of the mouths were decidedly unclean. All these patients with arrested caries were diabetic children who had been on a special diet for six months or more. This diet consisted of meat, eggs, butter, milk, vegetables, fruit, and cod-liver oil, with no sugars or other carbohydrates allowed.

Boyd and Drain attributed the arrest of caries in these cases to the correction of faulty nutrition and to the intake of an adequate amount of vitamins and mineral salts. In order to determine whether the fact that the children had diabetes and were being treated with insulin had anything to do with the control of dental caries, the investigators put four non-diabetic children with active, soft dental caries on a typical diabetic diet for a period of four months. During this time no progress of the carious process in the existing open cavities could be observed. After these four children had resumed their ordinary diet, two of them developed recurrent, active caries.

It is impossible to review adequately all the experimental work on the relationship between diet and dental caries in man. The above-mentioned studies of the Michigan group and of Boyd and Drain are only two of many similar studies. The importance of an adequate supply of vitamin D in the diet is emphasized, among others, by Mellanby and Agnew, Agnew and Tisdall, that of vitamin C by Howe and Hanke. The articles by Kesel (*Jour. Am. Dent. Assn.*, **19**, 903, 1932), Rosebury (*Arch. Path.*, **15**, 260, 1933), Bunting (*Jour. Am. Dent. Assn.*, **22**, 114, 1935), and Arnim (*Int. Jour. Orth. Oral Surg.*, **23**, 1045, 1937) contain a review of this subject and are recommended for collateral reading.

Oral Bacteria and Caries.—Of the large variety of microorganisms that are habitually present in the human oral cavity, some forms have been considered significant in connection with caries. Bunting and his associates have made a thorough study of *Lactobacillus acidophilus*. *Lactobacillus acidophilus* is a short rod that is pleomorphic, that is, it can appear in different forms. It actively ferments carbohydrates, thereby forming lactic acid, and is capable

of surviving in its own acid products. The members of the Michigan group reported finding *L. acidophilus* in practically every case of active decay; immune mouths were notably free of it, and when *L. acidophilus* was artificially introduced in such mouths, it disappeared within a short time.

There appears to be a definite correlation between the carbohydrate content of the diet, the presence of *L. acidophilus*, and the occurrence of dental caries. In mouths with rampant decay *L. acidophilus* disappeared when carbohydrates were eliminated from the diet, after which the carious process did not progress farther. Jay and his associates reported greatly increased acidophilus counts in 91 per cent of a group of children during experimental feeding of large quantities of candy; in several children this increase was from several thousand microorganisms per cubic centimeter of saliva to 100,000 or more. In 86 per cent of the children the acidophilus count dropped after the feeding of candy had been discontinued.

In Bunting's opinion the only significant difference thus far known between caries-free and caries-susceptible persons is the difference in the number of *L. acidophilus* organisms in the mouth. This difference is closely correlated to the carbohydrate content of the diet.

The results of Bunting and his collaborators are not accepted everywhere. Some bacteriologists have been unable to duplicate their results. Greening streptococci, which produce acid in carbohydrate media, have also been isolated from dental caries, and recently Tunnicliff and Hammond have shown that the organisms found in the smooth and rough colonies of streptococcus viridans from carious teeth resemble the cocci, bacilli, and "tortuous threads" described by Miller in the dentinal tubules (see p. 129).

Further bacteriological and clinical studies of dental caries will be necessary in order eventually to reconcile the differences of opinion concerning the morphology and pathogenicity of the microorganism associated with tooth decay.

Experimental Production of Caries in Animals.—Another type of experimental investigation deals with the production of caries in animals. It is possible to produce structural imperfections and, in some instances, typical caries in the teeth of animals by certain variations in their food, especially by a reduction of the mineral and vitamin content and by an increase in the amount of carbohydrates.

The molars of the albino rat are excellent material for the study of experimental caries because they are in many respects similar to

human molars (Shelling and Asher, Rosebury *et al.*). The rat is an omnivorous animal that can easily be kept in large numbers. Its molars are subject to occlusal and, less frequently, to approximal caries with all the histological characteristics that are found under similar conditions in human teeth. The occlusal caries of rats begins at the bottom of the fissures and follows the course of the lamellæ to the dentino-enamel junction (Fig. 78). As the decay spreads into

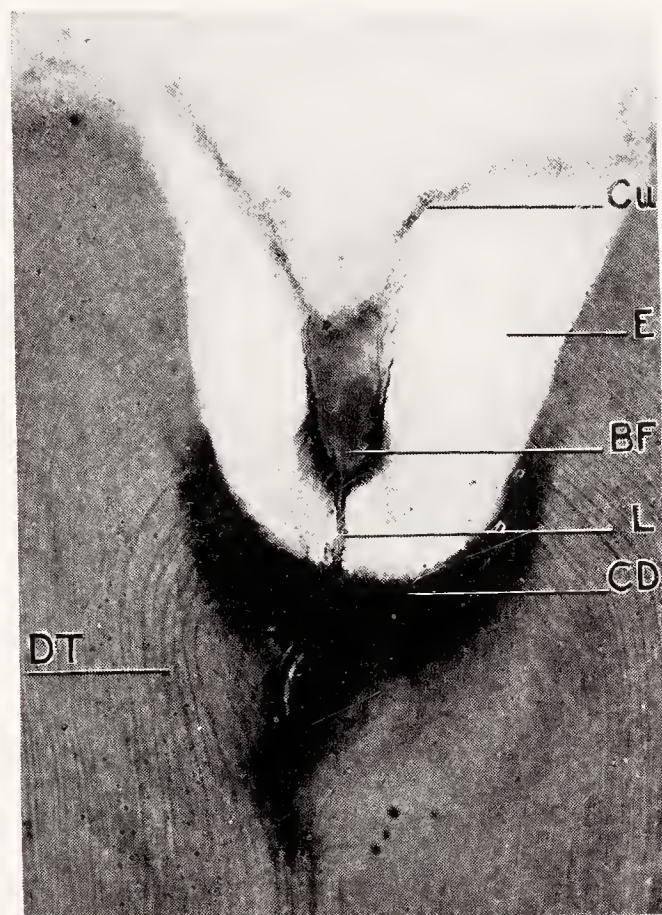


FIG. 78.—Incipient caries in an occlusal fissure of the molar of a white rat. The process of decay has passed from the bottom of the fissure through an enamel lamella to the dentino-enamel junction; from there it spread into the dentin, thereby undermining the enamel. Decalcified section. *BF*, bottom of occlusal fissure; *E*, enamel; *Cu*, cuticle; *L*, enamel lamella extending from the bottom of the fissure through the enamel into the dentin; *CD*, caries of the dentin; *DT*, infected dentinal tubules. (Barker, Jour. Am. Dent. Assn.)

the dentin, secondary dentin formation can be observed in the pulp chamber (Fig. 79). Eventually the pulp becomes exposed, pulpitis develops, and the final outcome is the complete destruction of the tooth with formation of a periapical abscess. Rats fed on a well-balanced diet are very little affected by caries, whereas animals kept on deficient diets show a high incidence of tooth decay.

CIVILIZATION AND DENTAL CARIES.

Dental caries is essentially a disease of civilization. That does not mean, however, that primitive and prehistoric peoples are or were altogether free of it; they, too, are and were affected to some

extent, but far less than modern man. The main difference between primitive and modern civilized man is their food. The transition from the primitive diet of meat, fish, milk, fruit, and coarsely ground corn or other cereals to the refined modern diet with its preponderance of finely ground white flour, sugar, and starchy foods is invariably accompanied by a rapid increase in dental caries, regardless of other external circumstances.

These statements are based upon abundant observation. Price found deterioration of the dental arches, malalignment of the teeth, and rampant dental caries in all parts of the world where the native population had adopted modern living habits and modern diet.



FIG. 79.—Ground section of rat molar showing caries produced by deficient diet. (Bunting.)

His field studies included such localities as Alaska, Australia and New Zealand, the South Sea Islands, and Africa. The figures he reported for caries incidence are very interesting. Among the Eskimos of western Alaska, living a very strenuous life under severe climatic conditions, the percentage of carious teeth was 0.09, or 1 tooth per 1000 teeth examined. Among the Eskimos living in areas where they had contact with modern civilization and imported foods, the incidence of caries was 13 per cent. Among the Indians of northern Canada the caries incidence was 0.16 per cent; among the Indian tribes living near the Hudson Bay Posts, it was 21.5 per cent. The incidence among the Melanesians (South Pacific Islanders) who were dependent on native foods was 0.38 per cent; among those who ate trade foods in considerable quantities, it was 29 per cent.

Waugh made similar observations among the Eskimos of Labrador and Alaska. Those that still live on their native diet of seal meat, fish, various smaller sea animals, and occasional native berries or plants are free of caries. The caries ratio increases in proportion to the amount of imported flour, sugar, and molasses that is consumed in a particular locality. Among the Eskimos, particularly among Eskimo children, living in the larger Alaskan cities, the food supply of which is not essentially different from that of New York or Chicago, the incidence of caries is about as high as among present-day American city dwellers. In addition, there is marked decrease in the size and strength of the dental arches, and malalignment of the teeth.

Roos reported an interesting caries study among the natives of Goms, a high, formerly almost inaccessible valley in the mountains of southern Switzerland. This valley had been inhabited by the same people for at least two thousand years; migrations, wars, plagues, and religious and political upheavals had apparently passed by without affecting this small isolated community. The inhabitants of the valley were farmers who lived on the produce from the native soil. Their staple foods were rye, barley, oats, beans, some green vegetables, the meat of cattle, goats, and sheep, and dairy products, such as milk, whey, and cheese. Wheat and fruit did not grow in the valley because of the high altitude. Sugar was practically unknown; it had to be brought in by porters over steep, narrow mountain paths and was considered an expensive luxury. The bread of coarsely ground whole rye was baked twice or three times a year in the individual farmhouses and then dried and stored. Under these circumstances the native population was practically free of caries, as can be seen in the numerous skulls found in the burial places of the valley.

Toward the end of the nineteenth century, a road and later a railroad were built, and stores were established that sold usual food products, wheat flour, sugar, potatoes, and other things that had not been available before in the valley. Bakeries were established which sold wheat bread. The accompanying deterioration of dental health was remarkable. Roos showed, by plotting on a map of the valley the percentage of caries among the school children in the various villages, that the incidence of tooth decay was in direct proportion to the nearness of the general stores and to the general accessibility of the village. In the least accessible mountain village the percentage of caries was 21; near the lower end of the valley, on the main road and railroad, the percentage was as high as 67.

In concluding this brief review of a few of the many observations concerning the correlation between caries and civilization, the author wishes to add a few comments of his own. If one studies the reports of Price, Waugh, or Roos, one cannot escape the question: What can be done about it? For the purpose of arresting dental caries should the present level of civilization or, rather, domestication be abandoned, with a reversal to the living and eating habits of an Alaskan Eskimo or a primitive Indian? Obviously this is not the solution to the problem. It seems that man will always have to pay in some way for additional comfort in his mode of living, and dental caries is one of the demands made in exchange for the domestication of our diet.

But at the same time it must not be forgotten that enormous progress has been made in many phases of medicine and public health which amply compensates for the increase in dental disease. The primitive inhabitants of Alaska or the South Sea Islands, or even the farmers in the secluded Swiss valley years ago were helpless in every major medical emergency. Appendicitis, diphtheria, complications in childbirth, extensive injuries meant almost certain death. The progress of transportation and civilization and the introduction of modern foodstuffs have no doubt been the primary cause for the increase in dental caries; however, by the same channels of communication, sanitation and medical aid have entered, and the incidence of disease and mortality has been greatly reduced. In the future, oral care, brushing of the teeth, and regular dental treatment will have to take the place of the former caries immunity.

CARIES OF THE ENAMEL.

Enamel Cuticle and Enamel Lamellæ.—The destruction of the dental hard tissues by caries starts in the crown portion of the tooth on the surface of the enamel. This is true of all caries except that of the cementum, which occurs in old individuals. The enamel is covered by the cuticle, which plays an important rôle in the early stages of dental decay since caries of the enamel develops only after the cuticle has been destroyed.

The enamel cuticle is continuous with the ends of the enamel lamellæ on the tooth surface. The lamellæ are bands of organic structure running perpendicularly from the enamel surface through the enamel to the dentino-enamel junction, or into the dentin. They are natural lines of weakness in the enamel along which caries can travel from the tooth surface into the enamel and to the dentin.

There are two kinds of enamel cuticle, the primary and the

secondary (Gottlieb). The primary cuticle is formed by the ameloblasts; it is very thin and usually calcified. It is easily dissolved by acids, and, therefore, it is of no significance in connection with dental caries. The secondary cuticle is thicker than the primary. It is formed by the cells of the epithelial attachment, and since in later periods of life the epithelium is attached to the surface of the root, the secondary cuticle can also be found on the cementum. It is resistant to dilute acids and alkalies; in decalcified teeth it is left after all of the enamel has disappeared.

The secondary cuticle is formed by that portion of the epithelial attachment that corresponds to the stratum corneum of the oral epithelium. It is the result of keratinization of the cells of the epithelial attachment. Thus, the hornified cuticle may be looked upon as a continuation of the protective keratinous layer of the oral epithelium over the erupted portion of the tooth.

The secondary cuticle is not always present. In mouths in which the gingival epithelium has little tendency toward formation of a horny layer, the secondary cuticle is usually poorly developed or entirely missing, while in other mouths it is present on every tooth. In erupted teeth it is found only in those areas of the tooth surface that are protected from wear during mastication, namely, on the gingival portion of the teeth, on the approximal surfaces, and in the bottom of the grooves and fissures. On all other parts of the tooth surface the cuticle is worn away.

In order to study cuticle and lamellæ, it is essential to use both ground and decalcified sections. In ground sections the enamel is preserved, but the non-calcified structures, especially the cuticle, cannot be clearly distinguished. In decalcified sections the enamel is lost, but cuticle and lamellæ are left; they can be stained and examined under the microscope. In ground sections prepared at right angles to the long axis of the teeth, lamellæ appear as dark lines running from the tooth surface into the enamel and sometimes into the dentin (Fig. 80). If a section in the same plane is made through a decalcified tooth, an entirely different picture is found. The enamel has disappeared, and the lamellæ have lost their support and have folded. By using special precautions during decalcification of the enamel and embedding of the specimens, it is possible to keep the cuticle in its original position after the loss of the enamel. Then the relationship between cuticle, lamellæ, and the dentinal part of the lamellæ can be studied.

The cuticle probably acts to some degree as a protection to the enamel surface against the action of acid-producing microorganisms. It covers the outer part of the enamel lamellæ where the latter reach

the enamel surface. Orban has shown that the hornification may extend into the peripheral portion of the lamella (Fig. 81). Under such circumstances the lamella is as protected against bacterial invasion as the rest of the tooth surface. On the other hand, teeth without a cuticle and without hornification of the peripheral ends of the lamellæ are, no doubt, less resistant to bacterial activity.

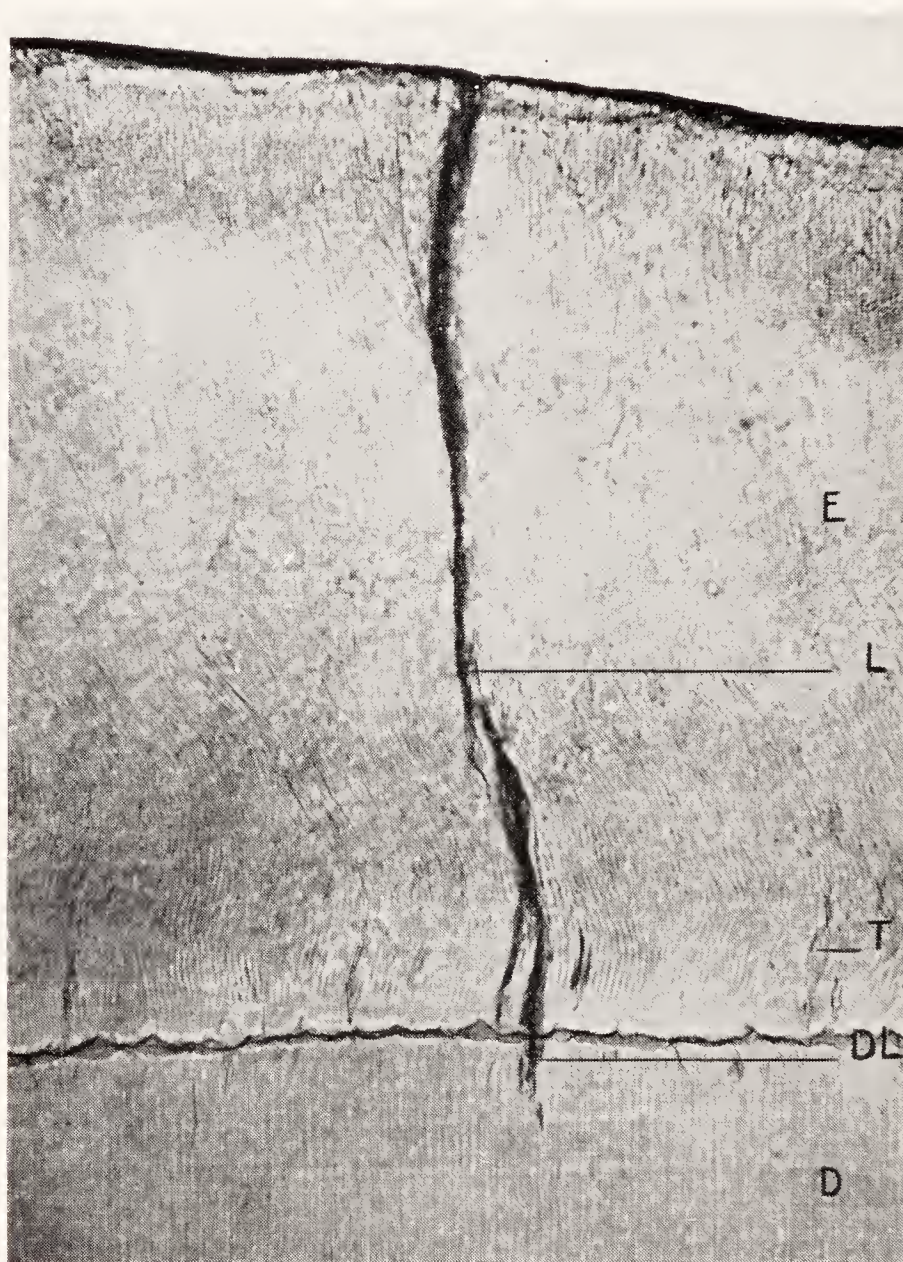


FIG. 80.—Enamel lamella extending into the dentin. Ground section. *E*, enamel; *D*, dentin; *L*, enamel lamella; *DL*, dentinal part of the lamella; *T*, tufts.

Thus, the cuticle may play a rôle in determining the susceptibility of a tooth or tooth surface to decay.

It is customary to distinguish between caries of the smooth enamel surfaces and caries of the grooves and fissures. The decay of the smooth enamel surfaces will be considered first.

Caries of the Smooth Surfaces of the Enamel.—Decay on smooth enamel surfaces occurs in the approximal and cervical portions of the teeth. The initial step in the development of caries in these locations is the formation of a plaque on the tooth surface. A plaque is an accumulation of gelatinous material on the tooth surface;

it consists of living and dead microorganisms, mucus, food débris, and desquamated and degenerated cells. Under ordinary circumstances a plaque is invisible; it is firmly attached to the enamel and can be removed only by scraping with a sharp instrument.

The formation and location of plaques are influenced by masticatory conditions, by the form and arrangement of the teeth, and by the amount of oral care. They normally never develop on the cusps and incisal edges, except in the case of enamel hypoplasia, since mastication prevents any accumulation of material in these areas. The favorite locations for plaques are the cervical portion of the crown, the approximal surfaces, and the depths of pits and fissures.

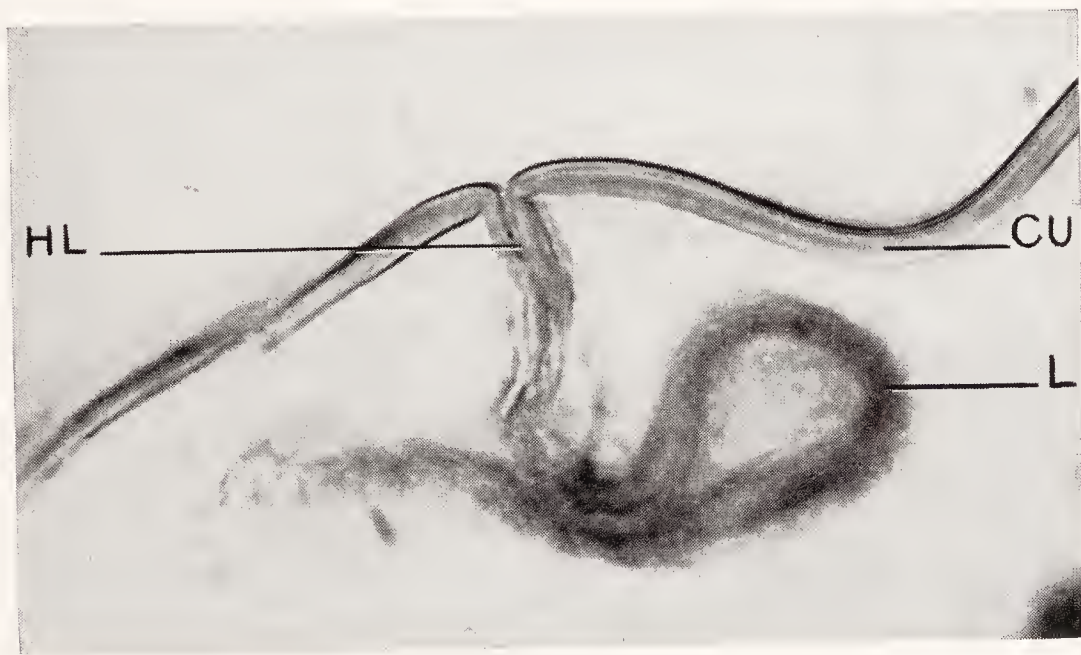


FIG. 81.—Hornified (secondary) cuticle and enamel lamella. The peripheral part of the lamella is formed by a duplication of the cuticle. Decalcified section. *CU*, cuticle; *HL*, hornified outer part of the lamella; *L*, inner part of the lamella, consisting of organic material without hornification. The lamella has been torn loose from the dentin and has folded after the decalcification of the enamel.

Here they can form without being disturbed by mastication or by brushing of the teeth.

A plaque may lead to decalcification and caries of the underlying enamel. Whether or not decay actually develops depends upon several factors. One of these is the presence and condition of the cuticle. A tooth with a well-developed cuticle apparently offers greater resistance to decay than a tooth without such a cuticle. Another important factor is time. Unless the plaque is allowed to remain undisturbed for a sufficient length of time, no decalcification of the underlying enamel can occur. This can be observed especially in children with poor oral care who have developed superficial decalcification of the enamel under large plaques. If these children are taught the correct and regular use of a hard tooth-brush, the decay may come to a standstill.

Very important is the presence or absence of carbohydrate residue derived from sugars and starches in the diet. If such fermentable material is lying on the tooth surface, the enamel soon becomes decalcified. Acid-producing microorganisms are apparently closely correlated with the presence of carbohydrates (see page 102).

The relationship between tooth surface, cuticle, and plaque is illustrated in Figure 82, which was taken from a horizontal section through a central incisor at a level slightly crownward from the bottom of the gingival crevice. The specimen was decalcified, which dissolved the enamel, and embedded in celloidin. Plaques are present on both the mesial and the distal side of the tooth,



FIG. 82.—Horizontal section through gingival portion of an upper central incisor. Decalcified section. *D*, dentin; *E*, space formerly occupied by the enamel; *BP*, bacterial plaque on the enamel surface; *CU*, hornified cuticle; *L'* and *L''*, enamel lamellæ torn loose from the dentin and folded.

except where the enamel was covered with a secondary cuticle. At intervals lamellæ extend from the tooth surface toward the dentin. The peripheral portion of some of them has been invaded by bacteria (Fig. 83). This condition may be interpreted as the earliest onset of caries; although neither enamel nor dentin is as yet actually decayed, masses of microorganisms are present at the peripheral ends of the lamellæ, ready to invade the enamel. The accumulation of bacteria in this area was apparently made possible or at least facilitated by the plaque on the enamel surface.

In a still higher magnification, the composition of the plaque can be recognized (Fig. 84). Most of it is an amorphous mass with occasional, faintly visible cell outlines. Near the tooth surface grows a colony of microorganisms forming long filaments; still closer to the enamel there is an accumulation of bacteria that have entered the peripheral part of an enamel lamella present in this area.

The early stages of caries on the smooth enamel surfaces can best be studied on ground sections through an area of discoloration (white or brown spot) or superficial destruction of the enamel. In such

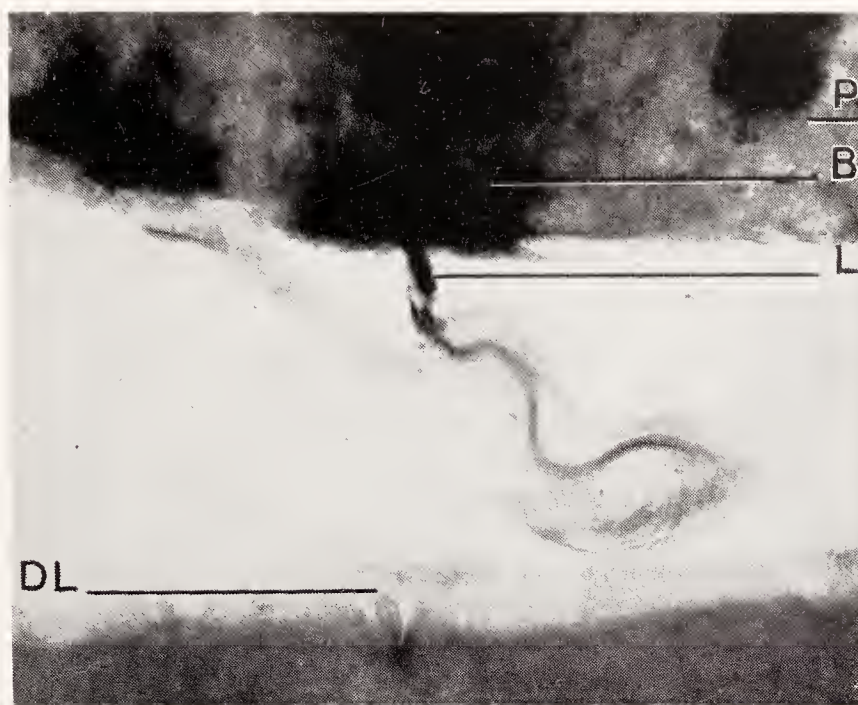


FIG. 83.—High magnification of a lamella under a plaque (Fig. 82, *L''*). *P*, plaque; *B*, accumulation of bacteria around the peripheral end of the lamella; *L*, peripheral portion of the lamella showing bacterial invasion; *DL*, attachment of the lamella to the dentin from which it was torn loose after the decalcification of the enamel.



FIG. 84.—High magnification of a lamella under a plaque. *M*, microorganisms forming long filaments on the enamel surface; *B*, bacteria on the enamel surface; *CU*, remnants of a hornified cuticle; *L*, lamella, the peripheral portion of which has been invaded by microorganisms.

sections the earliest carious lesions appear microscopically as superficial decalcification of the enamel without loss of substance on the enamel surface (Figs. 85 and 86).

In order to understand the changes occurring in enamel decalcification, it is necessary to consider the normal process of calcification

of the enamel. Enamel consists of prisms and interprismatic substance. Each prism or rod is formed by the union of numerous calcified globules or droplets (calcospherites) that are deposited

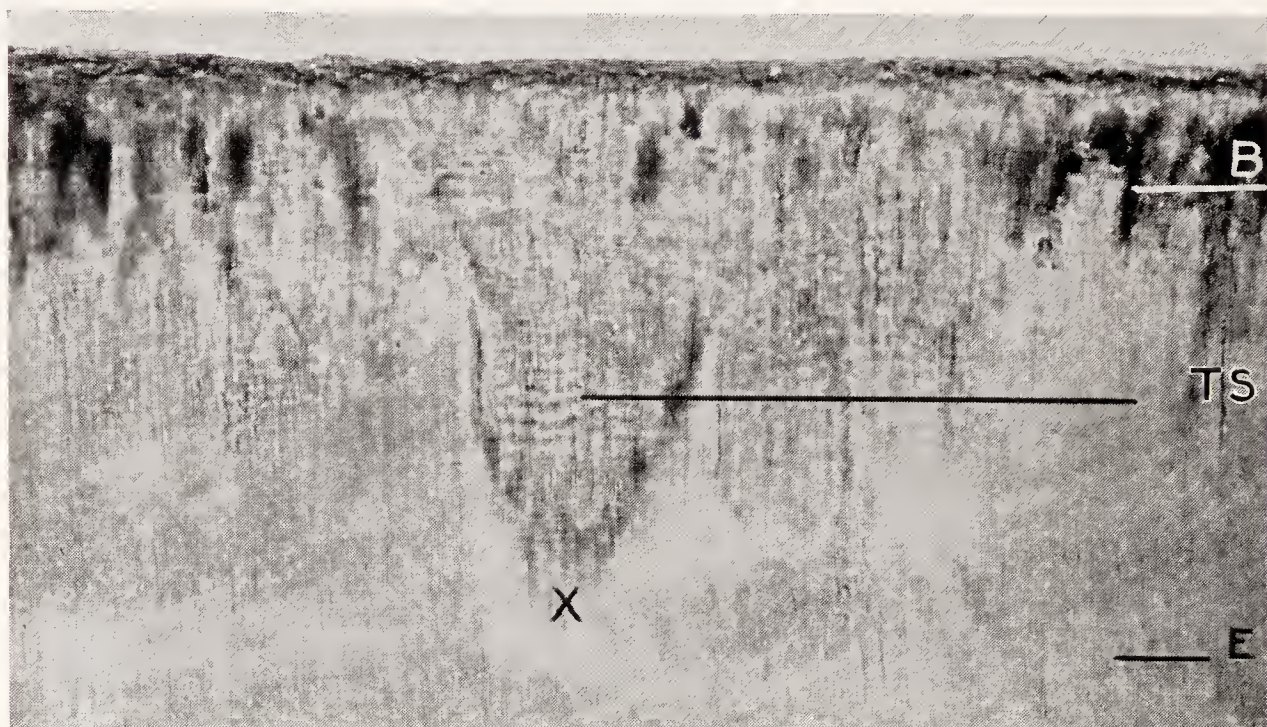


FIG. 85.—Superficial caries of the enamel on the labial surface of a tooth near the gingival margin (clinically a “white spot”). *B*, bacterial invasion of the enamel; *X*, deepest point of decalcification of the enamel; *TS*, transverse striation of the prisms at the borderline between normal and decalcified enamel; *E*, normal enamel.

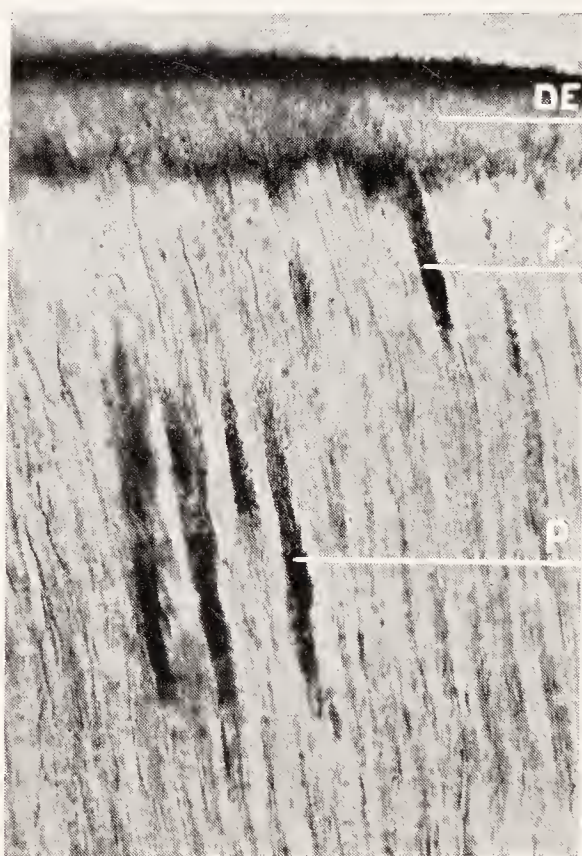


FIG. 86.—High magnification of Fig. 85. *DE*, superficial decalcification and disintegration of the enamel; *P*, decalcified and infected enamel prisms.

successively by the ameloblasts in the space inside the interprismatic substance or prism sheath. These globules calcify first; next follow the portions of the prism between the globules, and finally the inter-

prismatic substance. In decalcification the changes occur in reverse order: that part of the enamel that calcified last is decalcified first. Thus the interprismatic substance is decalcified first, followed by those portions of the prism that unite the individual calcospherites. As a result, these individual segments or divisions of each enamel prism become visible, giving the prism a segmented appearance; this phenomenon is called transverse striation, because the segmentation of each prism is indicated by dark lines running at right angles to the rods (Fig. 87). The same appearance of transverse striation can be observed when a ground section of intact enamel is exposed to the action of a dilute acid under the microscope; as soon as the acid reaches the enamel, the rods begin to show this

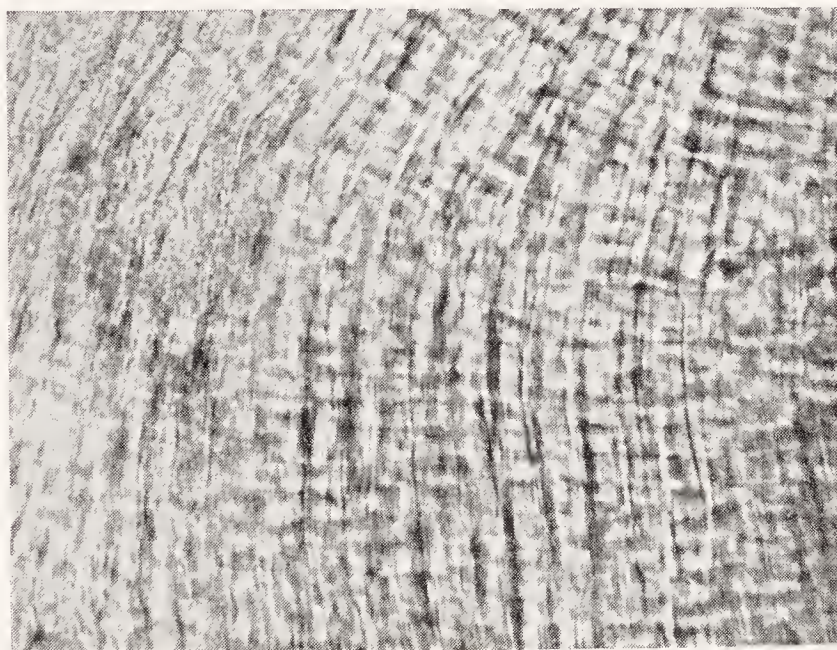


FIG. 87.—Transverse striation of enamel prisms in the periphery of a carious lesion of the enamel.

segmentation. Therefore, the assumption that such striation is the expression of the beginning of decalcification seems to be well justified.

Another change in connection with early caries of the enamel is the distinct appearance of the stripes of Retzius (Fig. 88), which are dark lines running more or less parallel to the enamel surface. They are also called incremental lines since they are the result of a rhythmic deposition or calcification of the enamel. Enamel calcification takes place by the precipitation of inorganic salts into a colloidal matrix. If the supply of these salts is abundant, the precipitation is continuous, and no stripes of Retzius are visible later. If, however, the supply is limited, zones of better calcification alternate with zones of poorer calcification; this rhythmic deposition finds its morphological expression in the stripes of Retzius.

During decalcification of the enamel by caries, the stripes of Retzius again become plainly visible; they are often found in the involved strata of enamel only, whereas they may be entirely absent in the deeper, intact layers of the enamel. Figure 88 shows these stripes in a very early stage of superficial decalcification of the enamel. This condition can hardly be called caries, although some loss of inorganic salts seems to have taken place.

Caries on a smooth surface of the tooth usually extends into the enamel in the form of a cone, the base of which is located on the enamel surface, with the tip pointing toward the dentin (Fig. 89). On the enamel surface bacterial invasion and the beginning of disintegration of the decalcified enamel prisms can be noticed; this is

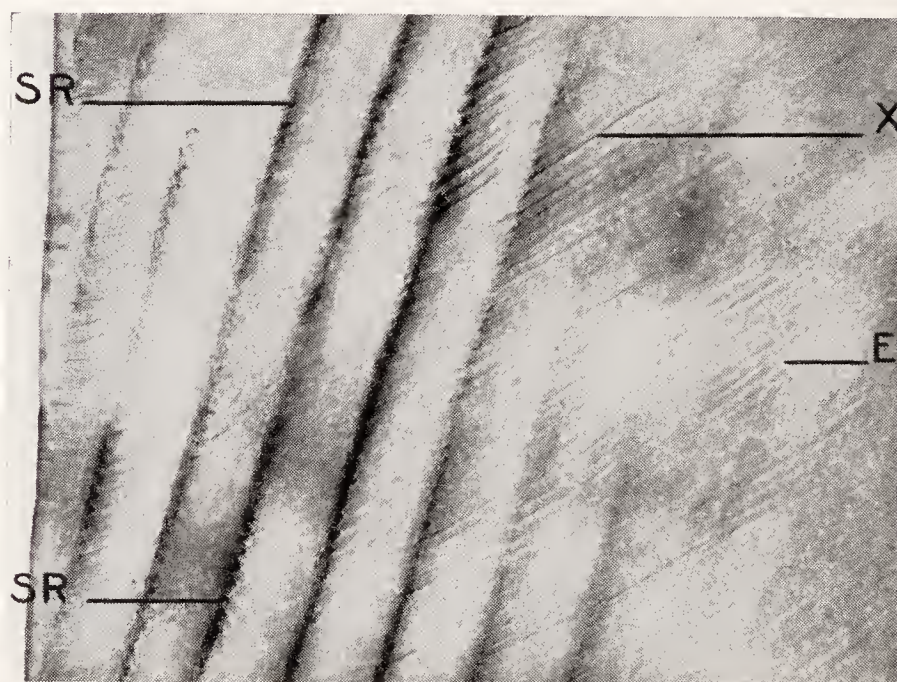


FIG. 88.—Marked stripes of Retzius in the superficial layer of enamel in the periphery of a “white spot.” *SR*, stripes of Retzius; *X*, incipient decalcification of the interprismatic substance in the enamel between the stripes of Retzius; *E*, normal enamel.

followed by a zone in which the rod structure is partly lost; then, next to the intact enamel, is a zone of transverse striation, an expression of decalcification that has just started. The stripes of Retzius are plainly visible in the affected enamel.

Applebaum studied the progress of demineralization of the enamel in incipient caries by means of soft roentgen-rays (Grenz rays). By comparing ordinary ground sections and radiographs of the same sections, the loss of minerals in the lesion could be demonstrated before there was an actual break in the surface continuity (Fig. 90). In the dentin the tract of sclerosed dentinal tubules below the carious lesion is more radiopaque than the surrounding normal dentin.

The conditions illustrated in Figures 85 to 90 correspond to what is clinically known as white or brown spots. There is enamel decal-

cification without loss of substance of the enamel surface. Whether such a decalcified area is white or brown depends largely upon the age of the patient and the rapidity of the destructive process. In

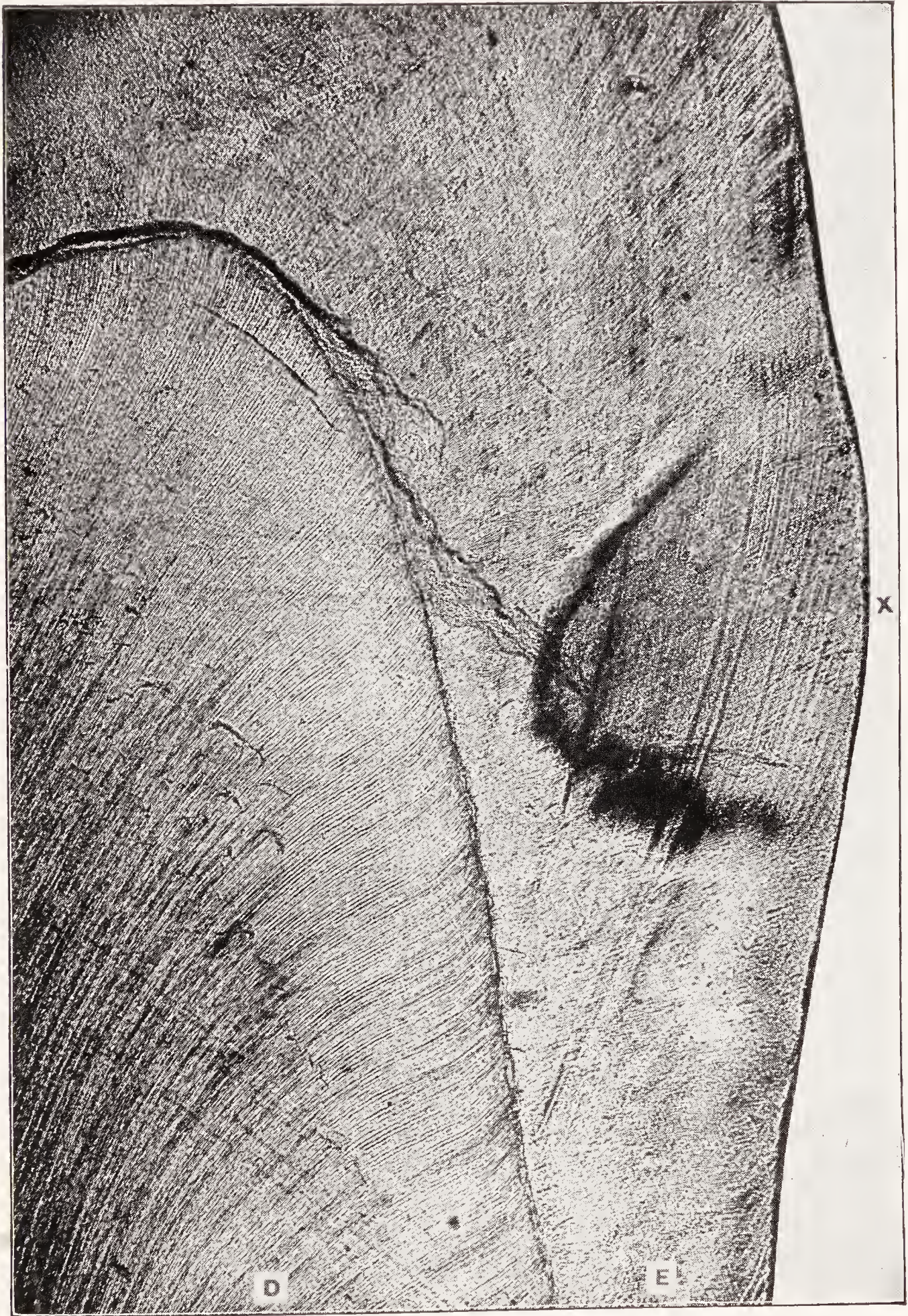


FIG. 89.—Section through a white spot in the first period of caries: *X*, disintegrated enamel; *E*, sound enamel; *D*, dentin. (Noyes-Schour-Noyes.)

the beginning, the decalcified spots appear white because the enamel has lost its normal translucency and has become opaque and chalky. Such areas of superficial decalcification may develop into true carious defects, or they may remain stationary for a long time, even for a lifetime. If the process of caries is slowed down considerably or



FIG. 90.—Unstained ground section of incipient interproximal caries of a human molar, with enlarged Grenz-ray photograph of the same lesion showing loss of density in enamel, evidently due to decalcification. (By courtesy of E. Applebaum and Dental Cosmos.)

completely arrested, the white spots gradually become brown, the result of pigmentation. The latter condition develops, for instance, after a tooth has been extracted, thereby exposing a carious mark on the approximating surface of an adjacent tooth. This lesion then lies in a self-cleansing area, the decay is arrested, and a brown spot develops.

By the time caries reaches the dentin, the enamel surface is usually already destroyed to such an extent that the tip of the exploring instrument encounters roughness or softness of the enamel caused by the loss of the superficial enamel rods (Fig. 91). If the surface of the decayed and softened enamel is examined under high magnification,

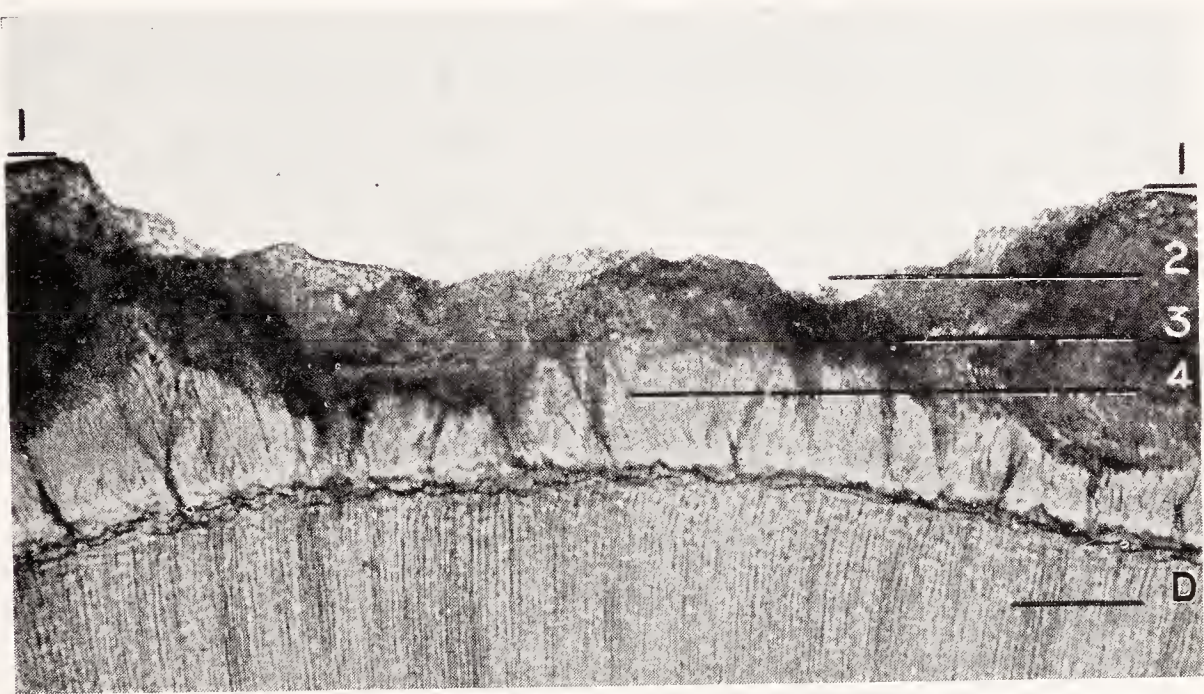


FIG. 91.—Caries of the enamel. Shallow defect in the enamel. 1, original enamel surface; 2, defect in the enamel; 3, dark brown pigmentation of the carious enamel; 4, enamel showing transverse striation; *D*, dentin.

the prisms appear separated because of the destruction of the interprismatic substance (Fig. 92); the peripheral ends of the prisms blend with the mass of bacteria and detritus overlying the carious enamel.

In summarizing the histological picture of active caries of the enamel, the following changes can be described in the enamel rods: near the surface of the enamel complete decalcification and softening of the prisms, in the deeper enamel layers beginning decalcification and loss of prism structure, and next to the dentin surface transverse striation of the enamel rods as earliest evidence of decalcification. However, these different stages cannot be found in every section, nor do they always occur in this order.

The location and progress of approximal caries of two molars are shown in Figures 93 and 94. In Figure 93 decay has just started

rootwise from the contact point. Both enamel surfaces are covered with plaques; the carious lesion involves the outer one-fourth of the thickness of the enamel. In Figure 94 the approximal decay is quite advanced. The contact point, however, is still intact. On the tooth to the left the cusp is extensively undermined. A tract of sclerosed dentin extends toward the pulp chamber. In the upper portion of the tooth to the right is a cone-shaped area of decalcification of the enamel; near the cemento-enamel junction a carious defect extends into the dentin.

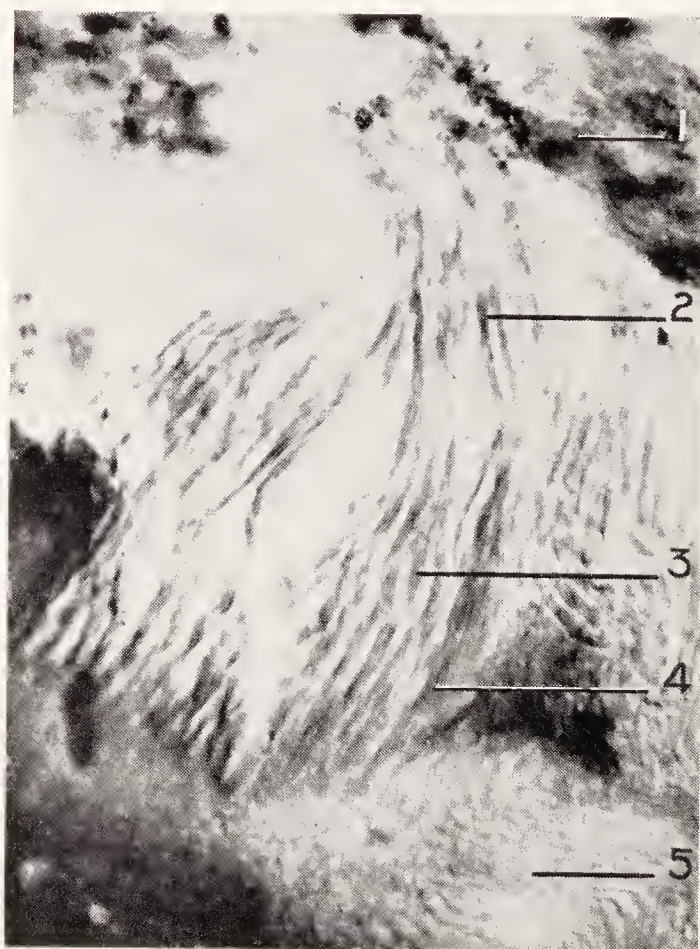


FIG. 92.—Decalcification and disintegration of the enamel prisms. Loss of interpismatic substance. 1, bacteria and detritus; 2, disintegration of the prisms; 3, enamel prisms separated by decalcification of the interpismatic substances; 4, transverse striation of the prisms; 5, intact enamel. (Decalcified section.)

Caries of the Grooves and Fissures.—Caries of the grooves and fissures occurs somewhat differently from caries of the smooth surfaces. The destruction spreads rapidly from the bottom of the fissure into the surrounding enamel and soon reaches the dentin, which is usually not far from the deepest point of the fissure.

The microscopic examination of the occlusal surfaces of normal human teeth reveals wide variations in the depth of the fissures. Some fissures are rather shallow, so that a thick layer of enamel separates the bottom of the fissure from the dentin. Other teeth have very deep fissures; then the bottom of the fissure is located almost at the dentino-enamel junction. Figure 95 shows such a

deep fissure in a lower first molar. The fissure is filled with detritus, but the surrounding walls of enamel are intact.

In occlusal caries decalcification of the enamel begins at the bottom of the fissure. From there it follows the general direction



FIG. 93.—Ground section through two adjacent molars showing approximal caries beginning rootwise of the contact point. (Bunting.)

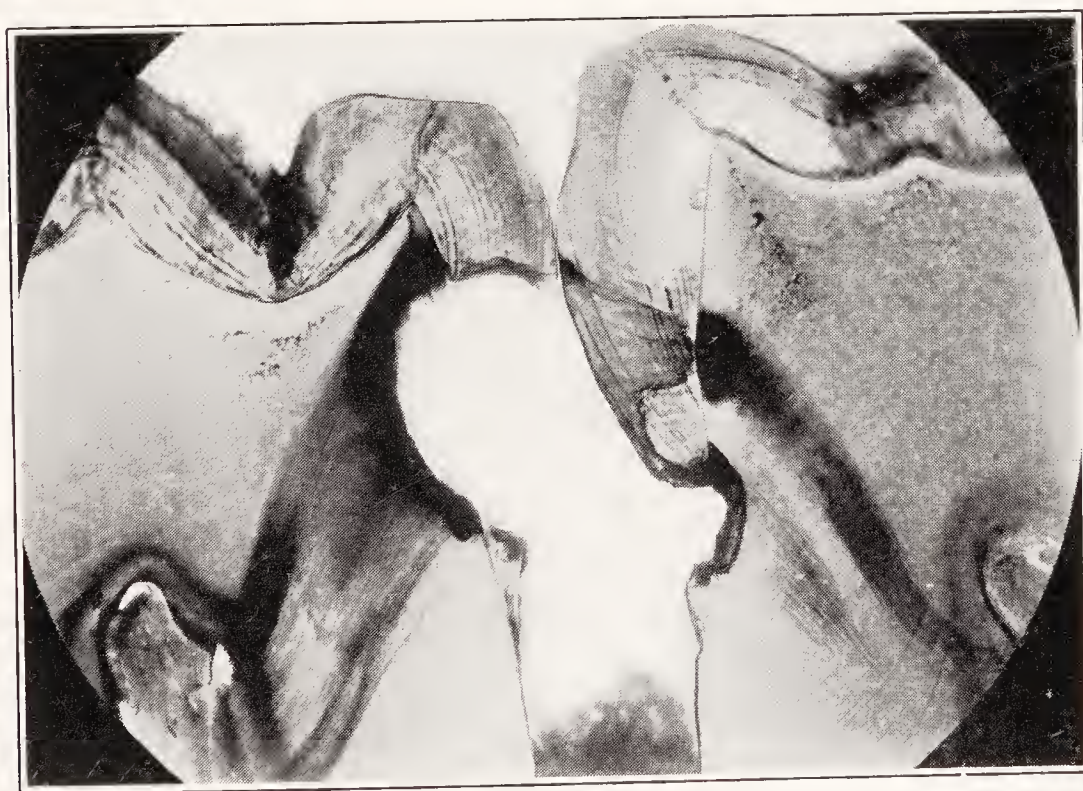


FIG. 94.—Ground section through two adjacent molars showing advanced approximal caries. The contact point is still intact. Note the tract of sclerosed dentin beneath the carious cavity. (Bunting.)

of the enamel rods. The changes that take place in the rods are the same as those in caries on a smooth enamel surface: complete dissolution of the rods at the bottom of the fissure, followed by a zone of decalcification of the interprismatic substance, then by a zone of

transverse striation, and finally by the normal enamel next to the dentino-enamel junction (Fig. 96). Since the enamel prisms diverge from the bottom of the fissure toward the dentino-enamel junction, the decayed enamel usually has the shape of a cone, the tip of which is located at the bottom of the fissure, the base at the dentino-enamel junction (Fig. 97). In addition, the decay spreads from the walls of the fissure so that sooner or later the superficial layers of enamel are undermined and lost, which results in widening of the

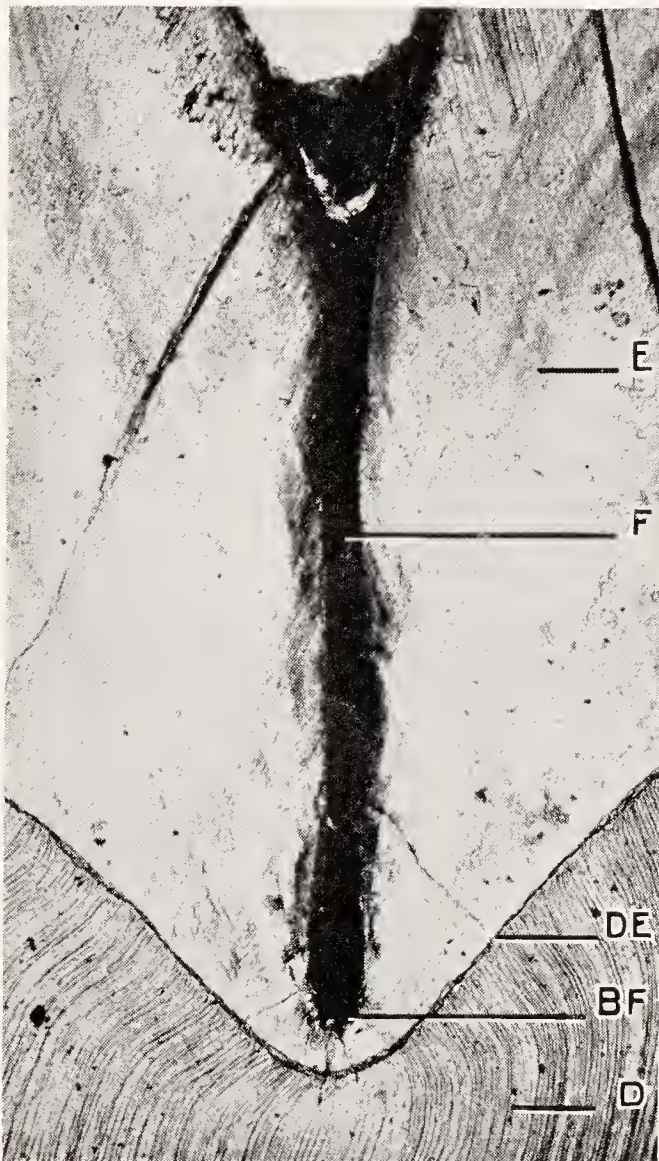


FIG. 95.—Deep intact occlusal fissure in a lower molar. *E*, enamel; *F*, fissure filled with detritus; *BF*, bottom of fissure; *DE*, dentino-enamel junction; *D*, dentin.

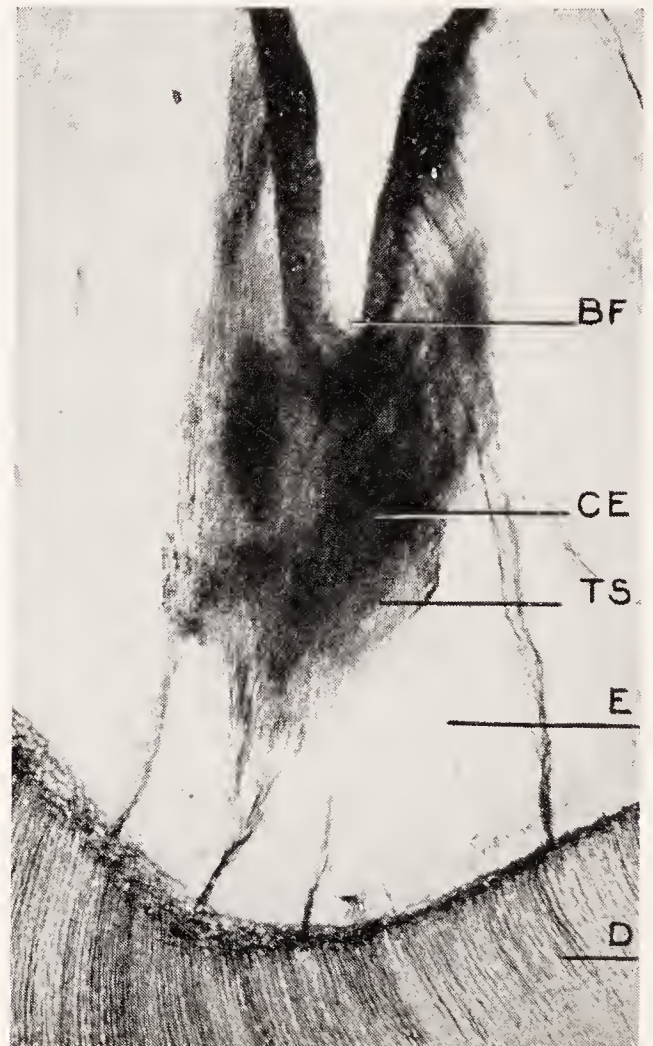


FIG. 96.—The beginning of caries of the enamel at the bottom of an occlusal fissure. *BF*, bottom of fissure; *CE*, caries of the enamel; *TS*, transverse striation of the enamel prisms; *E*, normal enamel; *D*, dentin.

fissure and roughening of the enamel margins (Fig. 98). Sometimes, however, the enamel in the occlusal portion of the fissure stays intact at first; the decalcification and destruction of the enamel spread for a considerable distance along the dentino-enamel junction, undermining the clinically healthy-appearing enamel around the opening of the fissure (Fig. 99). This explains the clinical observation that a fissure may, upon superficial examination, appear intact or only slightly discolored, although the occlusal enamel and dentin are extensively softened and decayed.

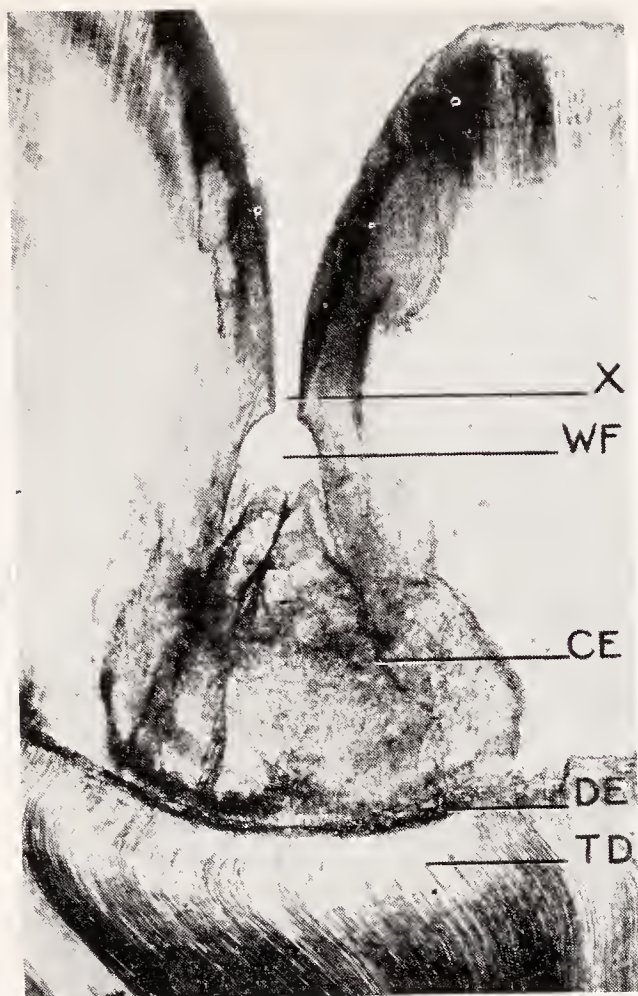


FIG. 97.—Caries at the bottom of an occlusal fissure. *X*, narrowest point of the fissure; *WF*, widening of the fissure caused by destruction of the enamel; *CE*, carious enamel at the bottom of the fissure; *DE*, caries reaching the dentino-enamel junction; *TD*, transparent dentin.

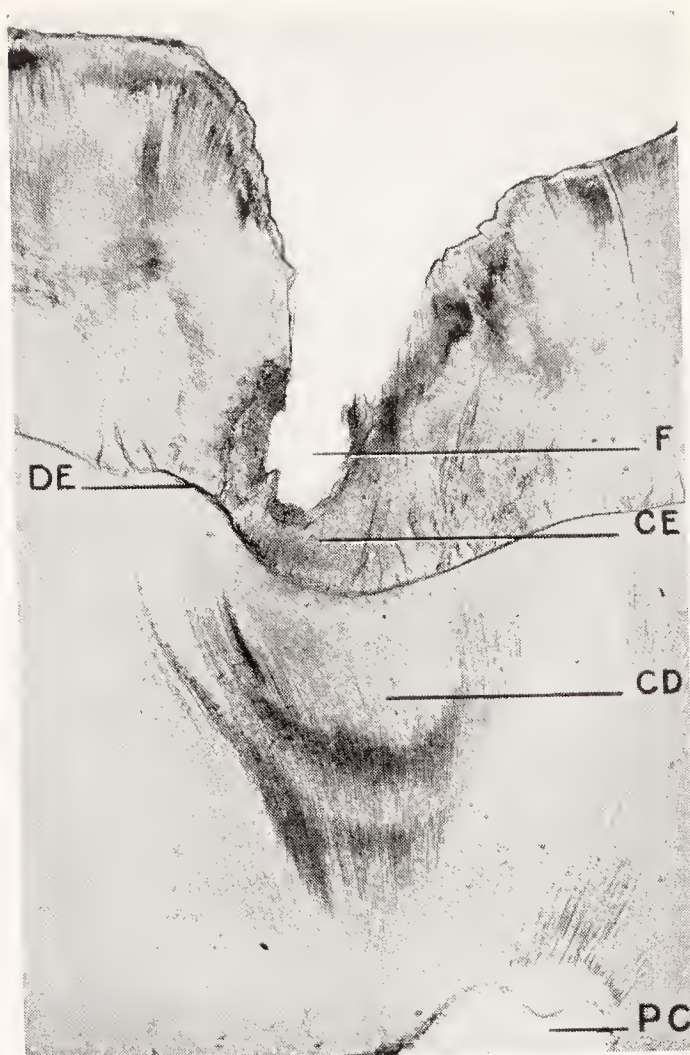


FIG. 98.—Advanced caries in an occlusal fissure of a lower molar. *F*, fissure widened by destruction of the enamel; *CE*, carious enamel at the bottom of the fissure; *DE*, dentino-enamel junction; *CD*, caries of the dentin; *PC*, pulp chamber.

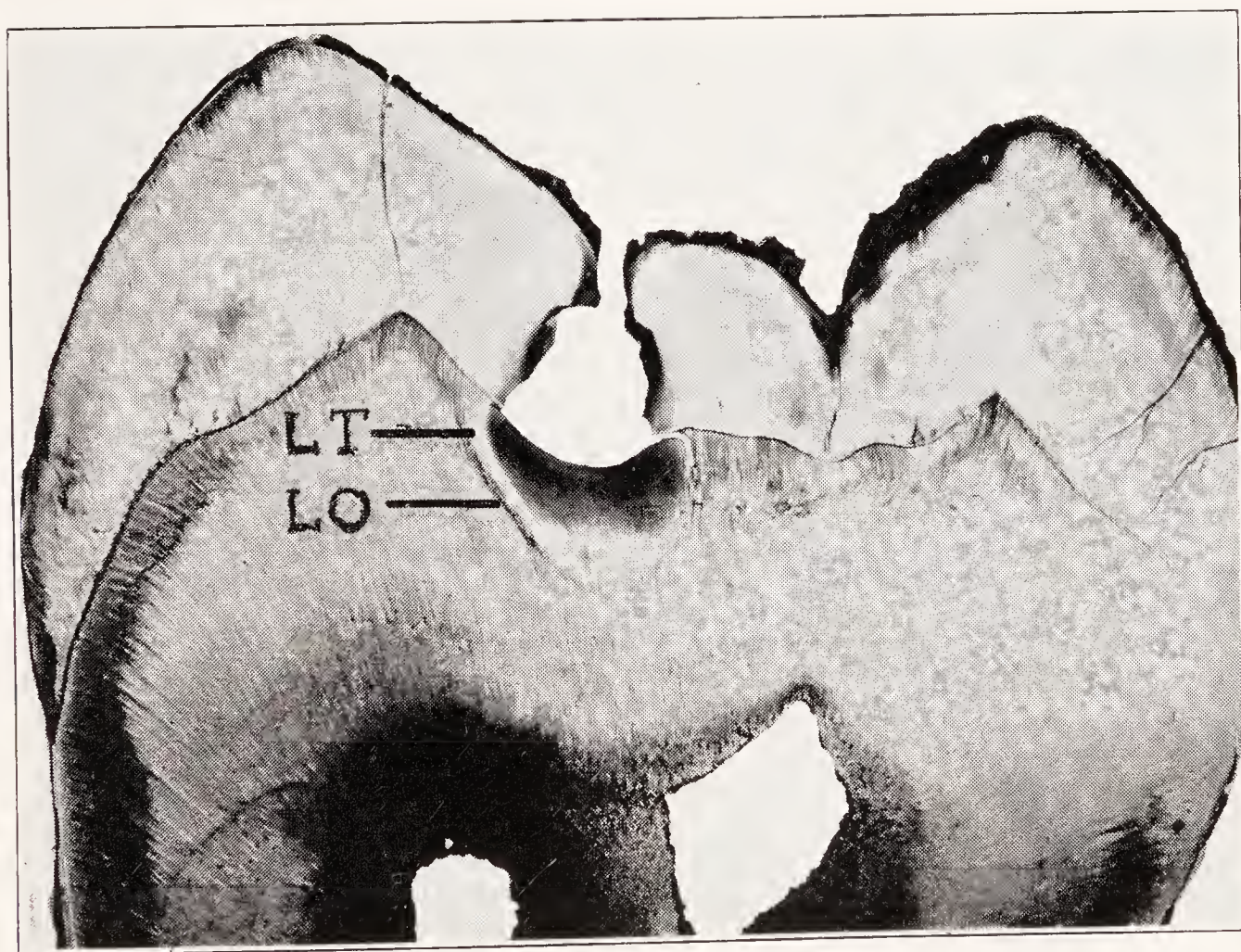


FIG. 99.—Ground section through a molar with the beginning of occlusal caries. Note the narrow defect in the fissure and the widening destruction toward the dentino-enamel junction. *LT*, translucent zone; *LO*, lateral opaque zone. (Beust, Jour. Am. Dent. Assn.)

Because of the rapid spread of decay along the dentino-enamel junction, the enamel may become decalcified from beneath (secondary caries of the enamel). This process accelerates the undermining and breaking-in of the non-supported enamel. Figure 100 shows extensive occlusal caries in a molar. The enamel of the occlusal surface is undermined, the decay having started from the dentino-enamel junction. Such unsupported enamel can easily collapse under masticatory pressure or under the pressure of an enamel chisel.

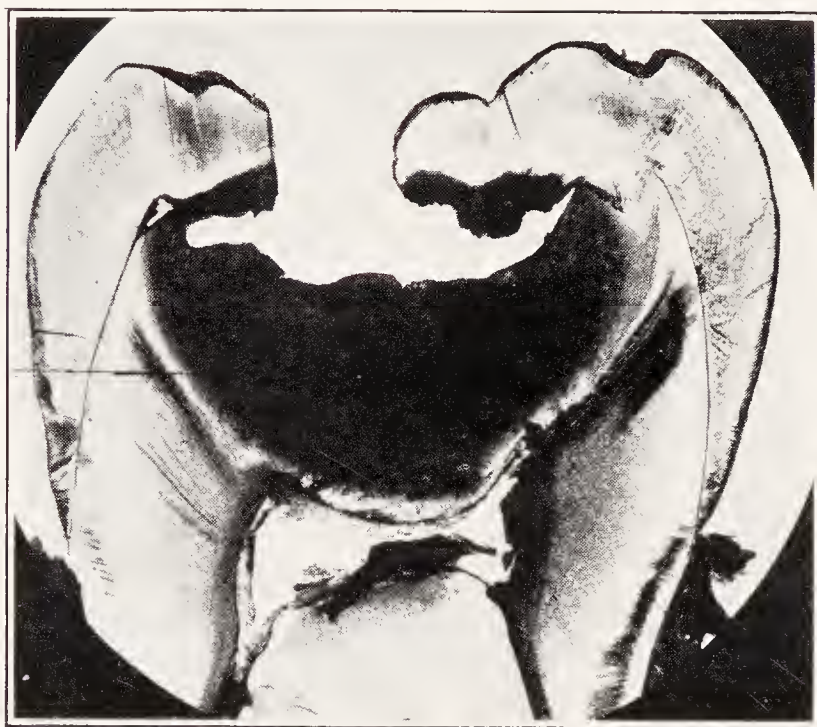


FIG. 100.—Extensive caries of the dentin, undermining the enamel and causing secondary caries on the lower surface of the enamel. (Bunting.)

CARIES OF THE DENTIN.

Significance of the Enamel Lamellæ in Caries of the Dentin.—In caries of the dentin, the dentinal tubules and their contents play an important rôle. They are natural pathways for the invasion and spread of microorganisms, and it is, therefore, not surprising that acute caries of the dentin runs a much more rapid course than caries of the enamel. On the other hand, the same structures that facilitate the spread of the decay, namely, 'Tomes' fibers, are capable of producing a calcified barrier as a means of defense. Thus, caries of the dentin in a tooth with a healthy pulp differs basically from caries of the enamel: the enamel has no reaction to the advancing microorganisms, whereas the living dentin reacts with a protective barrier.

Since the enamel lamellæ are of great importance in the onset of caries of the dentin, a few specimens illustrating lamellæ and dentin caries will be illustrated here, and in this connection the changes in the dentin will be discussed.

Figure 101 shows the relationship between enamel caries, lamella, and dentin. In the enamel the various zones that are characteristic of early carious lesions can be recognized: First, there is a structureless, brown surface layer, which is followed by a stratum of enamel that is invaded by microorganisms along the prisms. Nearer the dentin is a wide zone of transverse striation, the result of the penetration of acid into this part of the enamel. A lamella runs through the enamel and into the dentin. At the dentinal portion of this

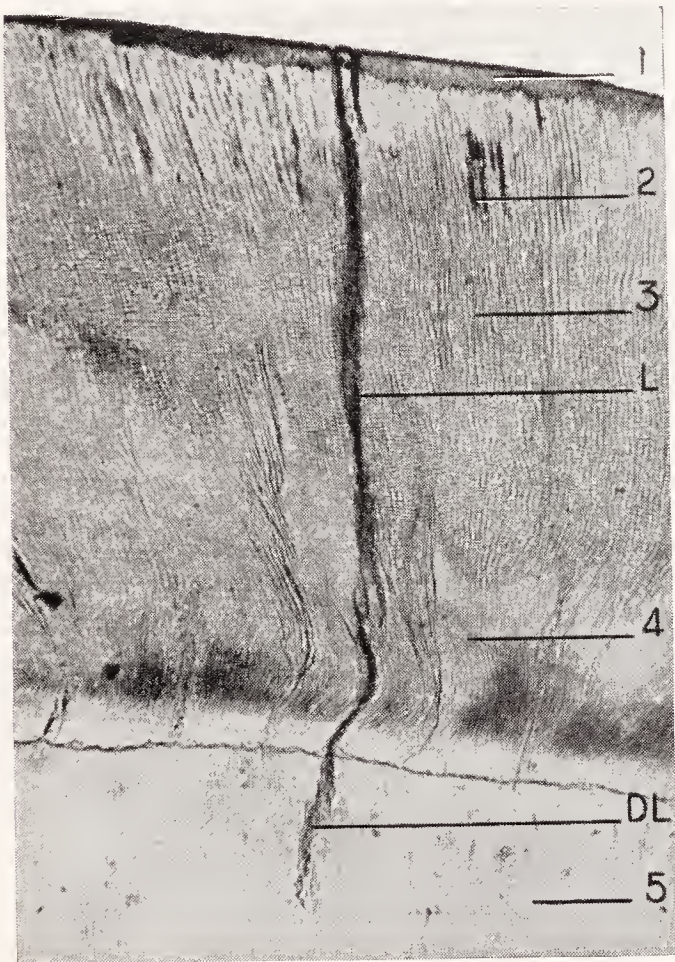


FIG. 101.—Transmission of enamel caries to the dentin by way of a lamella. 1, superficial structureless layer of carious enamel; 2, zone of bacterial invasion; 3, zone of transverse striation; 4, normal enamel; 5, dentin; *L*, enamel lamella; *DL*, dentinal part of the lamella.

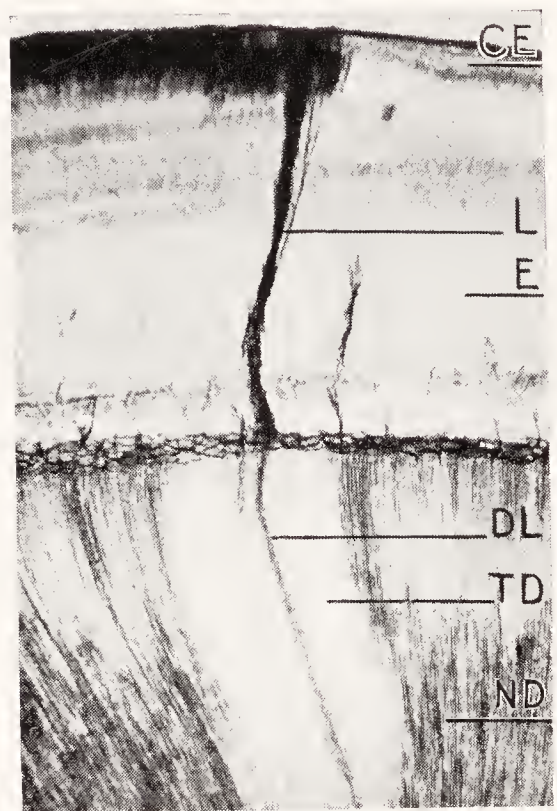


FIG. 102.—Transparent dentin around the dentinal part of an enamel lamella. *CE*, superficial caries of the enamel; *E*, normal enamel; *L*, enamel lamella; *DL*, dentinal part of the lamella; *TD*, transparent dentin; *ND*, normal dentin.

lamella the tubules are discolored, indicating beginning caries of the dentin. Probably microorganisms from the infected superficial enamel pass by way of the lamella through the deeper, intact enamel strata and reach the dentin, where they find much less resistance to their decalcifying action.

Early Stages of Caries of the Dentin.—The reaction of the dentin to the irritation transmitted by the enamel lamella is a condition known as transparency of the dentin (Fig. 102). The term transparency originated from the appearance of ground sections of dentin in which such areas appear lighter than the surrounding dentin. In

order to understand this phenomenon it is necessary to understand the normal optical appearance of dentin specimens. After dentin has been ground, treated with alcohol or xylene, and embedded in balsam, the tubules contain either air, remnants of Tomes' fibers, or balsam; or if the section has been stained, they contain a dye. Thus, the substance contained in the lumen of the tubule has a different refractive index than the surrounding dentin matrix; hence the tubule appears as a dark line in a light matrix (Fig. 103). If the dentin is irritated or injured, Tomes' fibers become calcified; the calcium salts that are normally in solution within the tubules are precipitated, and the tubules are obliterated by inorganic material. As a result, the

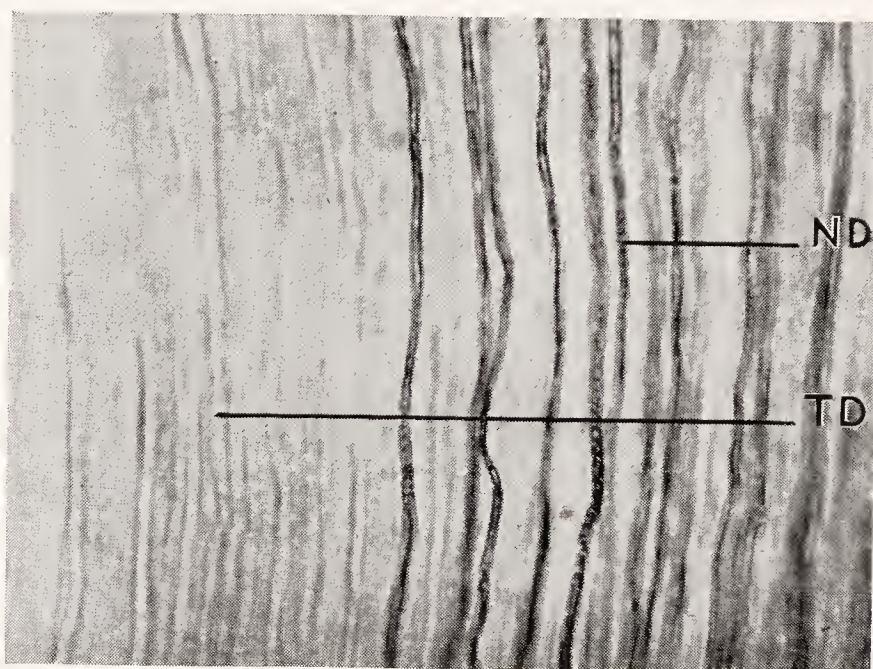


FIG. 103.—High magnification of the border between normal and transparent dentin in Fig. 102. *ND*, normal dentin; the tubules contain air and appear dark in transmitted light; *TD*, transparent dentin; the lumen of the tubules is filled with calcium salts. The tubules appear light in transmitted light.

difference between the optical refraction of the tubules and the matrix is greatly decreased and the dentin becomes transparent (Fig. 103, *TD*). Beust reported a difference in the penetration of dyes into the dentin under different conditions. Although the dentin of young teeth stained readily from the pulp chamber outward to the dentino-enamel junction, he found in older teeth areas into which the dye did not enter. Beust called these transparent areas sclerosed dentin, as they are the result of a precipitation of inorganic salts into the tubules. He found this change under abrasion as well as under caries; in either case the sclerosis is a defense reaction of Tomes' fibers to external irritation (see page 83). It is reasonable to assume that the slower progress of caries in the dentin of old teeth may be due to the increased calcification and decreased organic content of the tubules.

From this change in the dentin of teeth with vital pulps, Beust concluded that "an irritant acting on a fibril commonly causes a reaction within the tubule leading to its complete obliteration. Caries of the dentin, therefore, cannot be regarded as a simple proteolysis of dead tissue." In the dentin of a pulpless tooth caries is simply a decalcification and proteolysis; the bacteria enter the dead dentin and dissolve it without any defense reaction on the part of the tooth.

By staining ground sections with an alcoholic solution of diamond-fuchsin, Orban found that the dye entered all dentinal tubules and their ramifications except the zones of transparency, in which the tubules did not stain. This indicated that the latter were completely obstructed; their staining properties were not influenced by treating the section with ether, which suggested that they contained no fatty substance. The content of the tubules in the transparent area had the same optical properties as the dentin between the individual tubules.

A simple comparison may help to make the optical difference between normal and transparent dentin clear. If a glass tube containing air is submerged in water, it is plainly visible because of the difference in the optical properties of water and air. If the air is let out and water enters the glass tube, this difference is greatly decreased; the glass tube is then hardly visible. The air-containing tube corresponds to the normal dentin with non-calcified tubules; the tube containing water is like transparent dentin with calcified tubules.

Recent investigations concerning the changes in the dentinal tubules have revealed that the precipitation of calcium salts is not the earliest expression of irritation. If ground or frozen sections through transparent dentin are treated with fat stains (Sudan, scarlet red) fat droplets can be seen in the tubules (Euler and Meyer). This fatty degeneration of Tomes' fibers precedes the calcification of the tubular contents.

Once caries has involved the enamel, it is often transmitted to the dentin by way of an enamel lamella. In stained sections the invasion of the organic substance of the lamella can be plainly seen. Sometimes the enamel surrounding the lamella is slightly decalcified, showing transverse striation or discoloration; again, the enamel around the lamella may not be altered, the decay involving only the dentin at the point of attachment of the lamella. Figure 104 shows an early stage of caries of the dentin that was transmitted from the superficially decayed enamel by a lamella. On the surface the

enamel is brown and discolored, and in some places small defects are present. Slightly deeper the enamel shows transverse striation indicative of the beginning of decalcification; near the dentino-enamel junction the enamel is normal. At *L* a lamella runs through the enamel; at the point where the lamella reaches the dentin, caries spreads along the dentino-enamel junction and into the peripheral

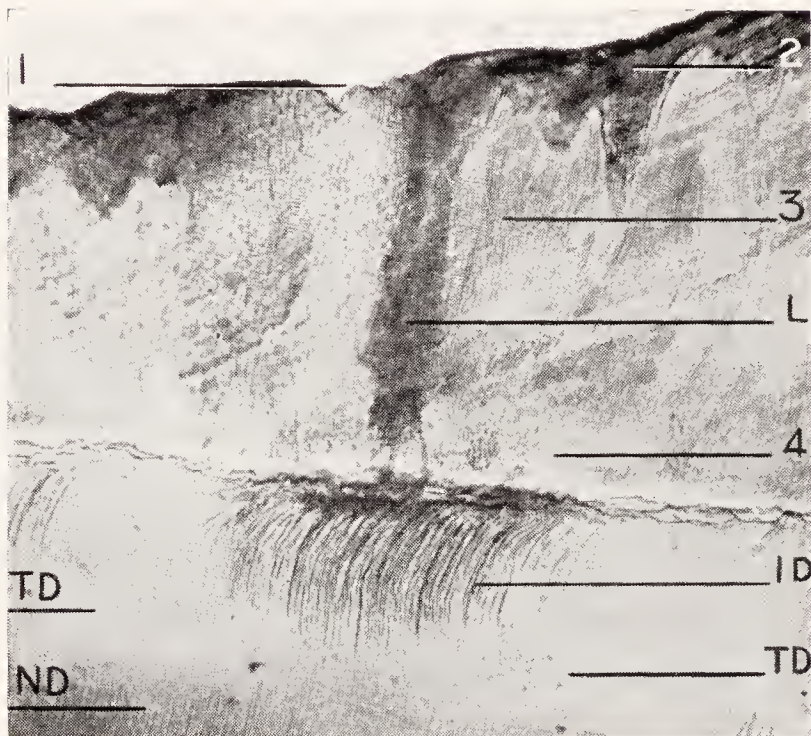


FIG. 104

FIG. 104.—The beginning of caries of the dentin, spreading from the dentinal end of an enamel lamella. 1, superficial defects in the enamel; 2, decalcification and discoloration of the enamel; 3, transverse striation; 4, normal enamel; *L*, enamel lamella; *ID*, infected dentinal tubules; *TD*, transparent dentin; *ND*, normal dentin.

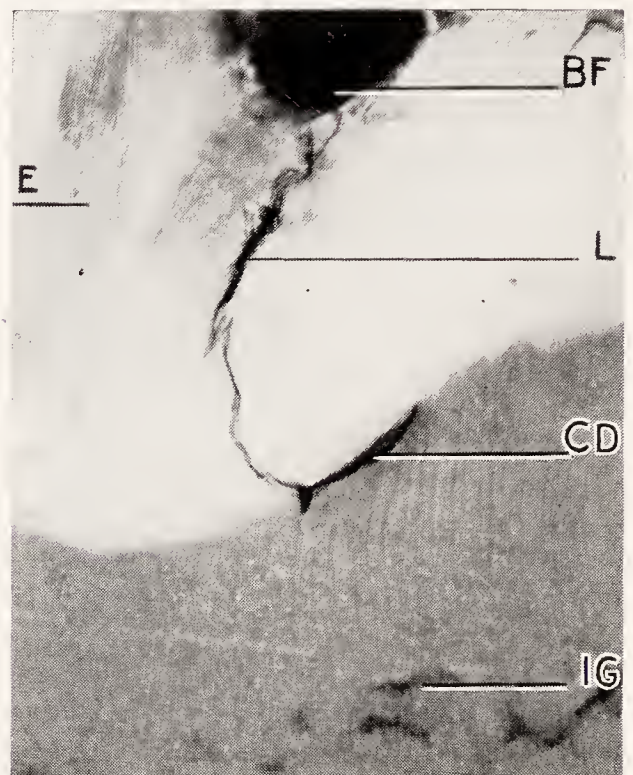


FIG. 105

FIG. 105.—Enamel lamella at the bottom of an occlusal fissure of a molar. Caries is transmitted through the lamella from the fissure to the dentino-enamel junction. Decalcified stained section. *BF*, bottom of occlusal fissure filled with detritus; *L*, enamel lamella; *E*, enamel; *CD*, caries of the dentin spreading along the dentino-enamel junction; *IG*, infected interglobular spaces. (Barker, Jour. Am. Dent. Assn.)

portion of the dentinal tubules. The infected tubules are surrounded by an area of transparency in the periphery of which normal tubules are visible.

In decalcified, embedded sections, the close relationship between lamellæ and dentin caries can be plainly seen. Figure 105 shows a lamella running from the bottom of an occlusal fissure to the dentino-enamel junction. The specimen was prepared from the lower first molar of a child; the enamel was lost, so that the lamella appears as a strand of organic tissue. Where this strand reaches the dentin, caries spreads along the dentino-enamel junction and into the adjacent dentinal tubules. The interglobular spaces in the dentin are also invaded by microorganisms. Barker has shown that in poorly calcified dentin the areas of uncalcified matrix between the calcified

globules are easily attacked by caries and offer but little resistance to the spread of infection through the dentin.

The first step in caries of the dentin is the invasion of the tubules by microorganisms; next follows the decalcification of the surrounding matrix by the action of these microorganisms. The finer details of the bacterial invasion and decalcification of the dentin are shown in later illustrations; first, however, a change will be described that is of great practical importance, namely, the retraction of the decalcified dentin from the inner surface of the enamel and the formation of spaces between enamel and dentin.

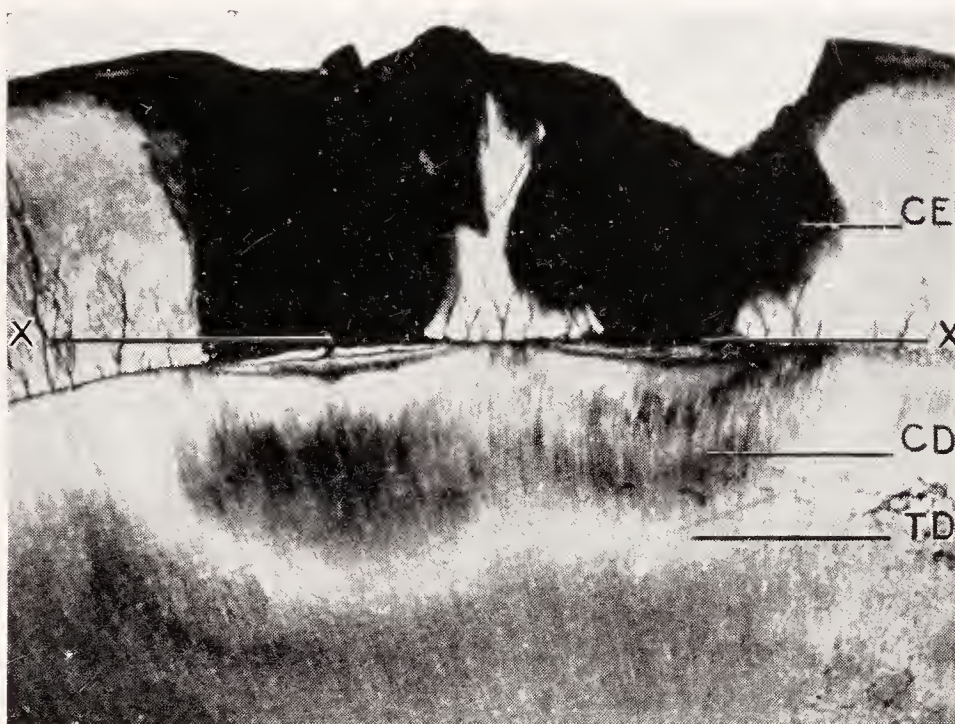


FIG. 106.—Caries of enamel and dentin. Shrinkage of the decalcified dentin; the dentin has retracted from the lower surface of the enamel, thus causing the formation of gaps between enamel and dentin. *CE*, caries of the enamel; *CD*, cariou dentin; *X*, gap between enamel and dentin; *TD*, transparent dentin. (Courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)

The decalcification of the dentin matrix causes a reduction in the volume and shrinkage of the matrix. The direction of shrinkage is at right angles to the dentin surface, parallel to the course of the tubules. Since the overlying enamel is less affected than the dentin, cavities or spaces with biconvex walls form along the dentino-enamel junction (Fig. 106). The presence of these spaces accounts for the ease with which the enamel overlying decayed dentin can be chipped with a chisel or enamel hatchet. This enamel is deprived of its support, and, therefore, it does not take much pressure before it will give way.

As caries penetrates into deeper layers of the dentin, the same changes in the tubules can be observed that were originally found near the dentin surface. Figure 107 shows a portion of a stained

ground section through carious dentin; on the left side of this illustration, dark masses of decomposed dentin cover the surface of the carious cavity. Next are visible dentinal tubules, which appear dark brown because of the presence of microorganisms. The next zone in the direction of the pulp is one of transparency or sclerosis, indicating a precipitation of calcium salts in the tubules. Finally, to the right, normal tubules are visible, appearing black because of air that entered during grinding. These various zones are especially distinct in the specimen illustrated in Figure 108, which is a stained ground section through a deep, occlusal, carious lesion in a molar

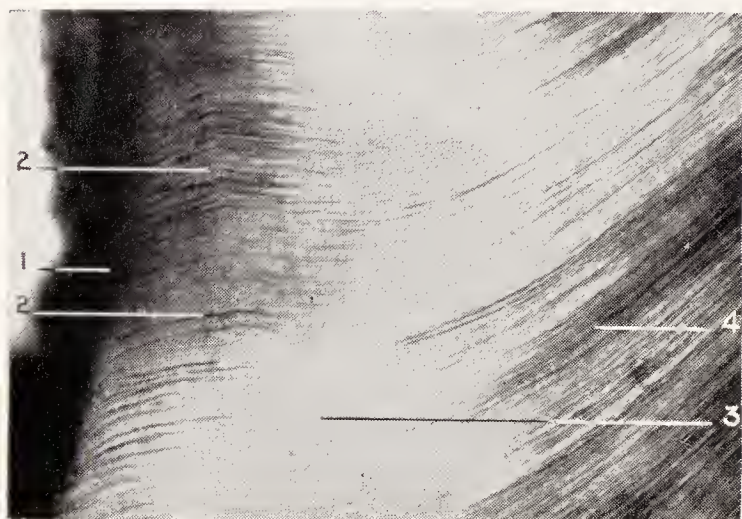


FIG. 107.—Caries of the dentin. Stained ground section. 1, structureless, decomposed dentin at the bottom of the carious cavity; 2, dentinal tubules invaded by microorganisms; 3, zone of transparent dentin; 4, normal dentinal tubules.

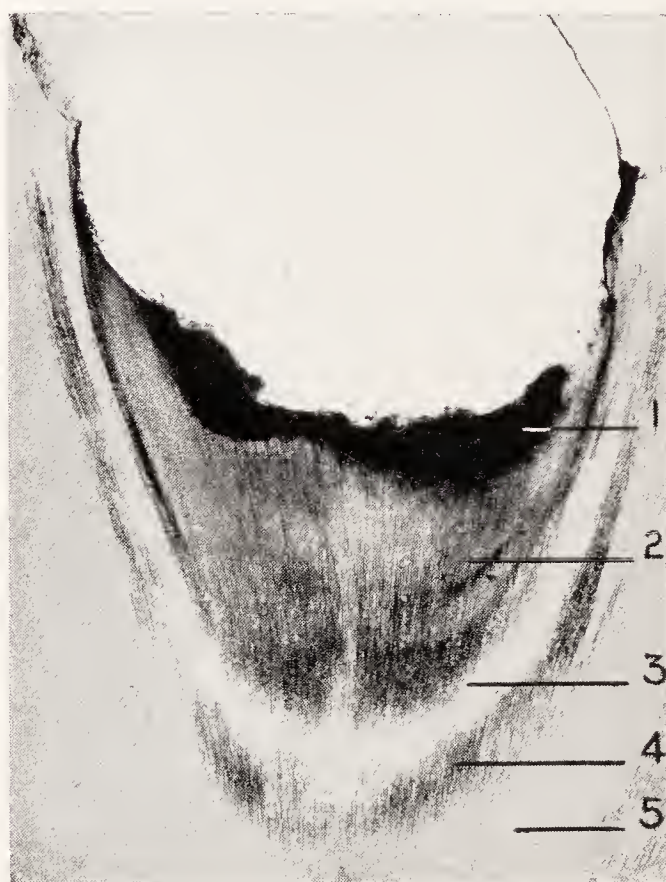


FIG. 108.—Caries of the dentin. Stained ground section showing the various zones in the dentin. 1, decomposed, softened dentin at the bottom of the carious cavity; 2, zone of decalcification of the tubules and invasion by microorganisms; 3, zone of transparency; 4, zone of fatty degeneration of Tomes' fibers; 5, normal dentin.

tooth. The dentin in the periphery of the decayed area is normal (Fig. 108, 5). Nearer toward the surface the dentinal tubules appear darker (Fig. 108, 4); this zone corresponds in its position and arrangement to the zone of fatty degeneration of Tomes' fibers. This zone is followed by a distinct, light area of transparency that encircles all of the decayed dentin (Fig. 108, 3). The latter appears dark, since the bacteria and the decomposed content of the tubules assume a dark purple or brownish color in a stained section (Fig. 108, 2). The cavity itself is lined by decomposed dentin (Fig. 108, 1). The study of such sections reveals that at least five distinct zones can be differentiated in the dentinal tubules of carious dentin. They are:

1. A zone of complete decalcification of the walls of the tubules and of the matrix with decomposition of the decalcified matrix.

2. A zone of incipient decalcification of the dentin, the result of the action of microorganisms that have invaded the tubules.

3. A zone of obliteration of the tubules by calcification of Tomes' fibers (zone of transparency or sclerosis).

4. A zone of fatty degeneration and the beginning of precipitation of calcium droplets in the protoplasm of Tomes' fibers.

5. A zone of normal dentin with apparently undisturbed dentinal tubules.

Zones 3 and 4 are caused by the disturbed metabolism of Tomes' fibers as a reaction to an approaching irritation; zones 1 and 2 are caused by bacterial invasion and decalcification of the dentin.

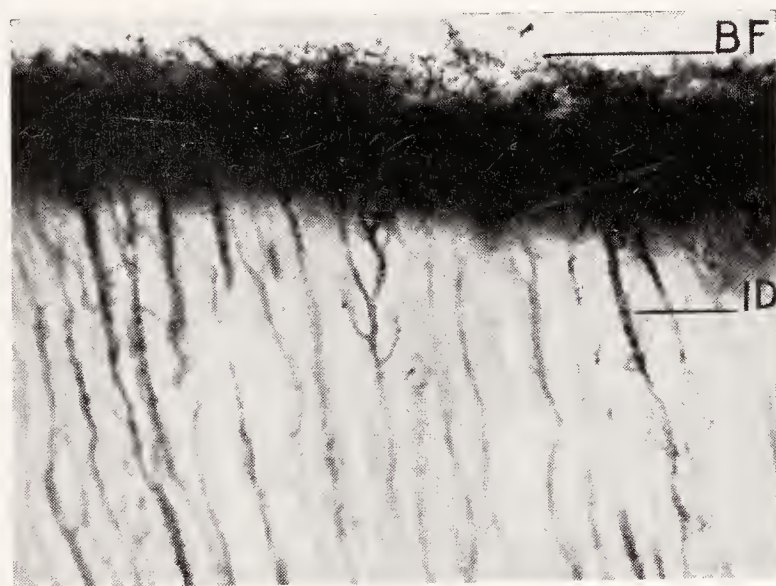


FIG. 109.—Bacteria invading the dentinal tubules at the dentino-enamel junction. *BF*, bacterial film on the dentin surface; *ID*, infected dentinal tubules and ramifications.

The minute changes associated with infection of the dentin can best be studied in decalcified sections through dentin stained with Gram stain. In such sections there is a marked contrast between those tubules that have been invaded by microorganisms and the surrounding, non-infected tubules. Figure 109 shows a section through a small, carious lesion at the dentino-enamel junction. The enamel has been destroyed; the surface of the dentin is covered with microorganisms that have begun to enter the dentinal tubules. The tubules and their terminal ramifications appear dark against the light matrix. In the deeper layers of dentin bacteria can be seen in some of the tubules, while others appear to be still normal.

Bacteria in Carious Dentin.—The morphology of the microorganisms in dentinal tubules has been known for over fifty years, ever since the fundamental studies of Miller were published. He

described three types of organisms, namely, cocci, bacilli (rods), and filaments (threads). Each of these has a tendency to grow by itself in individual tubules, although all three forms can usually be found in the same tooth in closely adjacent tubules of the decayed dentin. Only on the dentin surface, where the dentin is extensively destroyed, are the three types of organisms found mixed.

Figures 110, 111, and 112 show different dentinal tubules containing microorganisms. All these tubules, as well as the area shown

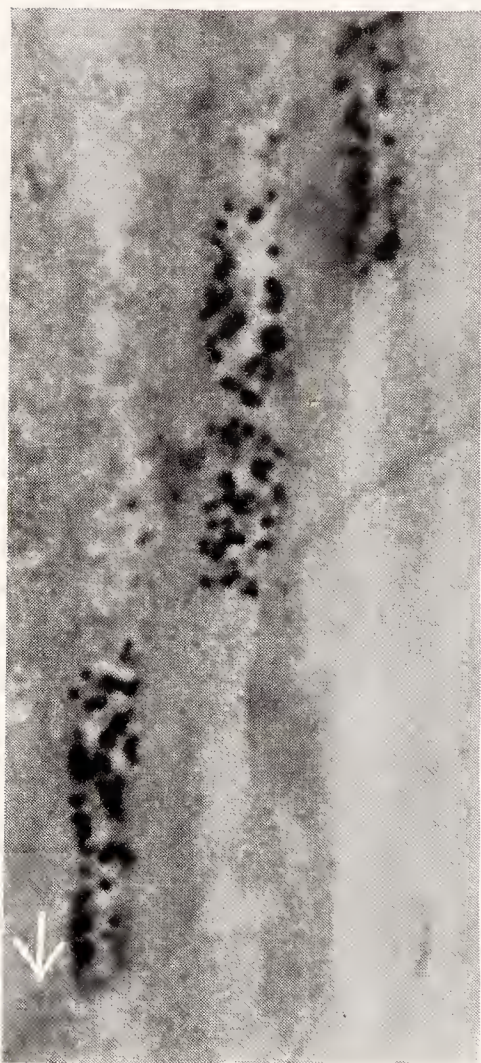


FIG. 110



FIG. 111



FIG. 112

FIGS. 110, 111 and 112.—Microorganisms in adjacent dentinal tubules of the same carious tooth (Gram stain). Magnification $\times 900$.

FIG. 110.—Three tubules containing groups of cocci.

FIG. 111.—Three tubules containing a large number of short rods (bacilli).

FIG. 112.—Two tubules containing tortuous filaments. (Courtesy of W. H. G. Logan.)

in Figure 113, were found in Gram-stained celloidin sections through a deeply decayed upper third molar of a woman, twenty-seven years old. In each illustration the upper part shows that part of the tooth nearest the carious surface, and the lower, that nearest the pulp. The arrow indicates the direction of growth of the organisms. The tubules in Figure 110 contain cocci that have progressed to slightly different levels in the adjacent tubules. In Figure 111 the tubules contain bacilli, and in Figure 112 long, tortuous threads or fila-

ments. Figure 113 shows an area of the decayed dentin surface in which both cocci and filamentous forms thrive.

The significance of these various forms is not yet fully understood. All three apparently produce acid and destroy dentin since distention of the tubules and liquefaction of the dentin similar to that



FIG. 113.—Microorganisms on the surface of decayed dentin. Same specimen as shown in Figs. 110, 111 and 112. Cocci and filaments. Gram stain. Magnification $\times 900$. (Courtesy of W. H. G. Logan.)



FIG. 114.—The beginning of distention of the infected tubules by the growth of bacteria and by decalcification of the surrounding dentin matrix. Beaded appearance of the infected tubules.

shown in Figure 114 are observed with each type. Whether the bacilli are lactobacilli, whether they and the cocci and filaments are separate organisms or are different forms of the *Streptococcus viridans*, as suggested by Tunncliffe and Hammond, cannot at present be stated with certainty. The fact remains, however, that

these three fundamental forms, or slight modifications of them, can be seen in nearly every well-stained section of decayed human dentin.

In studying the distribution of microorganisms in carious dentin, one is impressed with the fact that there are always a few tubules that contain organisms in a level far ahead of actual decalcification and decay. These "pioneer organisms," as they are sometimes called, are no doubt left in the tooth after all the decayed and infected dentin has been completely excavated during cavity preparation. However, clinical experience has shown that if such a properly prepared cavity is filled, no further decay occurs. Apparently the bacteria at the bottom of the cavity can no longer survive after the cavity has been tightly sealed with a filling. However, since there may be microorganisms in the tubules at the bottom of the cavity, the application of a dentin disinfectant, such as mercury bichloride, before insertion of a filling appears justified.

Advanced Changes in Carious Dentin.—The acid-forming microorganisms in the dentinal tubules decalcify the matrix that forms the walls of the tubules; this decalcified matrix is then destroyed, probably by proteolytic ferments produced by the same or different bacteria. In some of the tubules the bacteria, by their growth, distend the tubule at the expense of the surrounding softened matrix (Fig. 114); then the tubules assume a beaded appearance. As dentin decalcification and destruction progress, the matrix between the distended tubules is completely destroyed and transformed into a soft, crumbly mass; oblong cavities form parallel to the direction of the tubules. They are the result of the breaking-down of the matrix and the coalescence of a number of infected tubules. In Figure 115 this process of cavity formation in carious dentin is illustrated; on both sides, many infected tubules can be seen. The tubules coalesce and, by dilatation of their walls, form a large cavity that is filled with broken-down matrix and bacteria. That such cavities actually grow by expansion rather than by dissolution of the walls is indicated by the condition of the tubules in the surrounding decalcified dentin. The tubules in the periphery of the cavity deviate from their normal straight course; at the same time their width is reduced by compression. Both deviation and compression of the tubules are symptoms that the dentin in this area has been decalcified and is of soft, cartilaginous consistency. One would be tempted to speak of this condition as an abscess of the dentin, were it not for the fact that no cellular elements (pus) are present in the cavity, only necrotic dentinal matrix and bacteria.

Eventually the individual areas of disintegrating dentin coalesce, and all of the dentin is transformed into a soft, structureless mass. In a photograph of decayed, infected dentin under high magnifica-

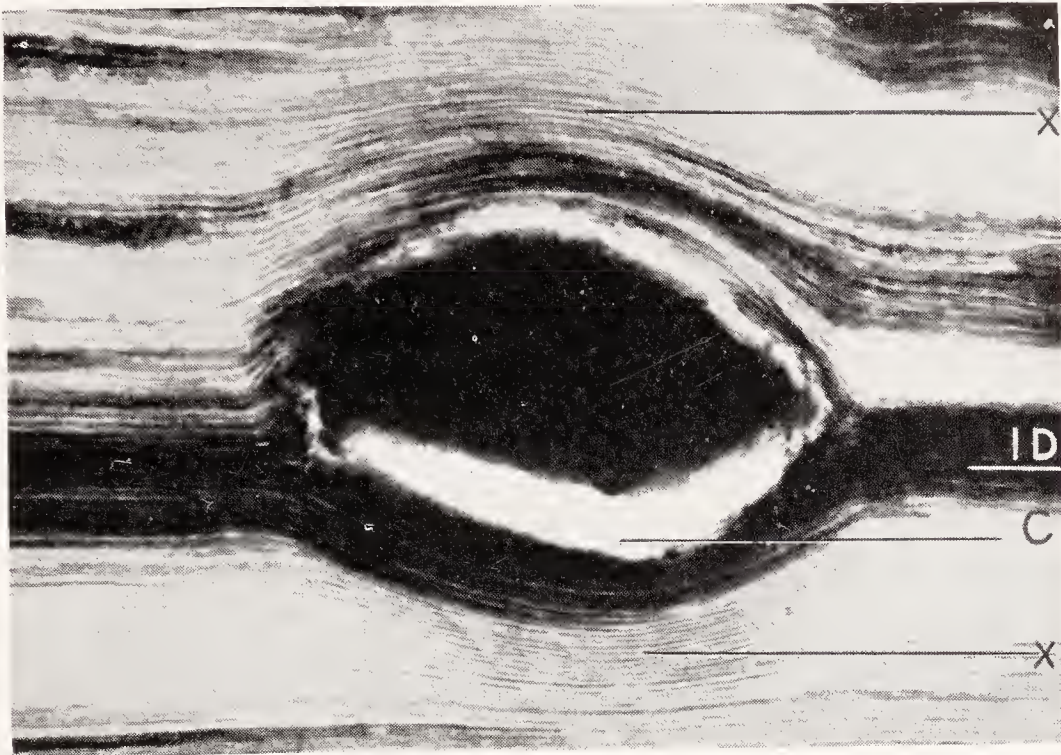


FIG. 115.—Cavity in the dentin formed by coalescence of infected dentinal tubules. The cavity contains detritus and bacteria. *C*, cavity; *ID*, infected dentinal tubules; *X*, deviation and compression of the tubules in the decalcified dentin in the periphery of the cavity.

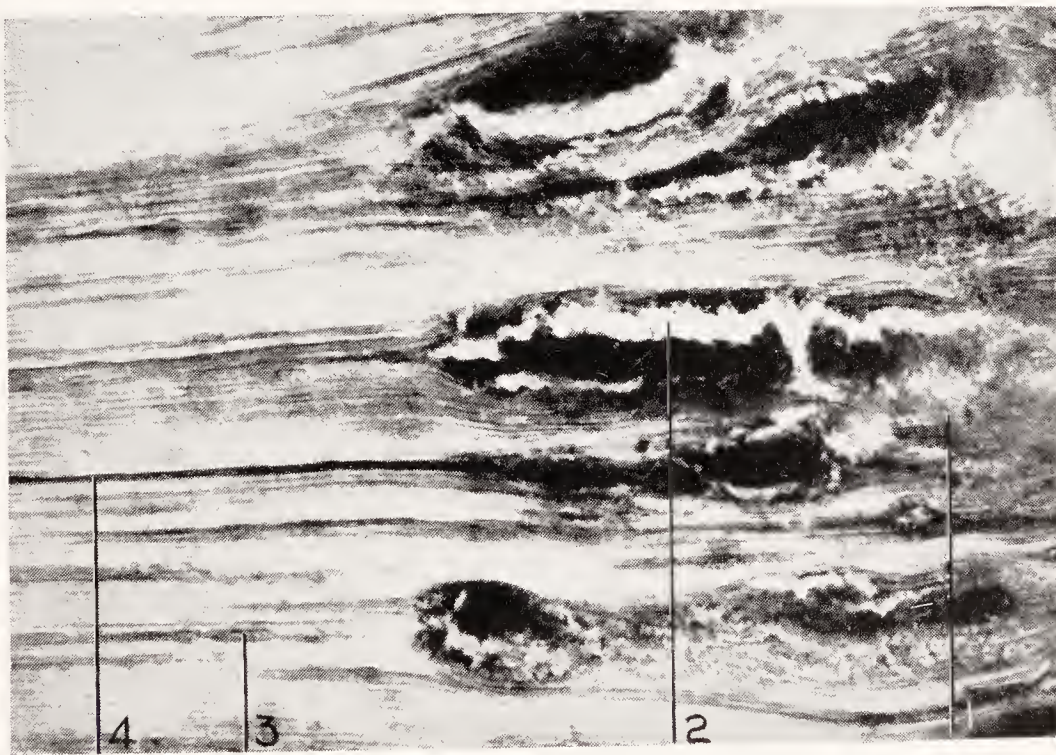


FIG. 116.—Advanced decomposition of carious dentin. 1, disintegration of the decalcified matrix; 2, formation of cavities by coalescence of the infected tubules; 3, swelling and distention of the infected tubules; softening of the surrounding matrix; 4, infected dentinal tubules.

tion, this transformation can be plainly seen (Fig. 116). On the left side of the illustration, the dentin contains numerous infected tubules. Close to the surface these tubules coalesce, forming large,

necrotic cavities. The most superficial dentin is completely decomposed and structureless. The carious surface is covered with a dense film of microorganisms, probably saprophytes, which digest and remove the necrotic dentin remnants (Fig. 117).

In many specimens of caries of the dentin, cavities or clefts can be observed that run at right angles to the course of the dentinal tubules (Fig. 118). This arrangement results from the course of the fibrils of the matrix. The dentinal matrix is built of fine fibrils that run

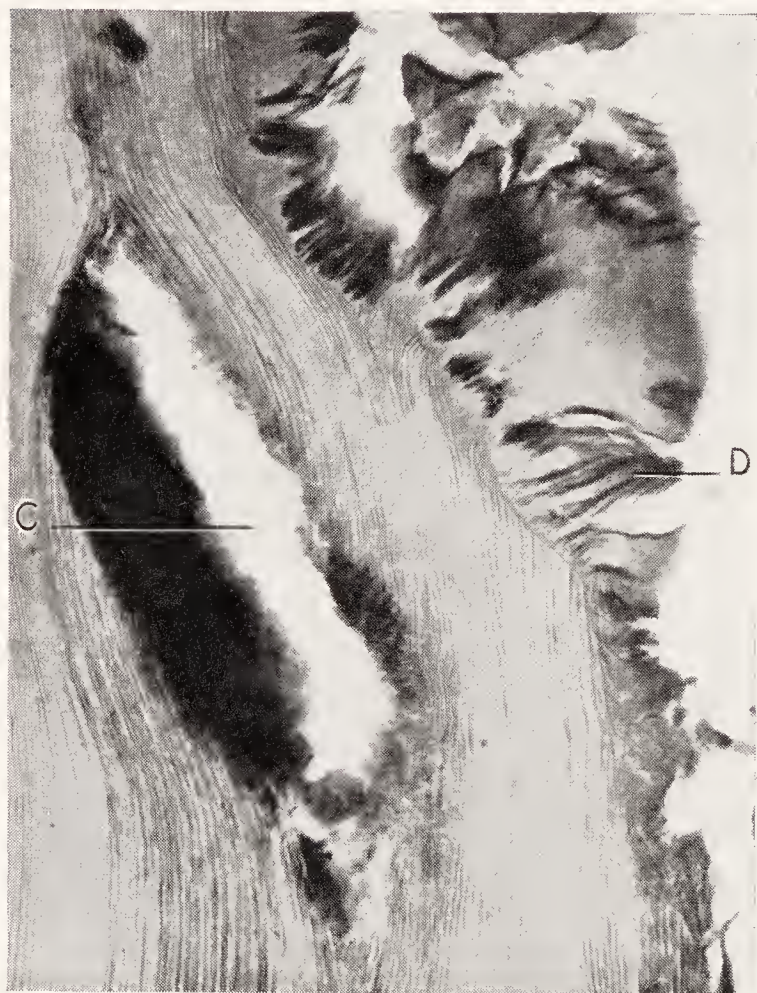


FIG. 117.—Superficial layer of carious and decomposing dentin. *C*, large necrotic cavity in the softened dentin, causing compression and deviation of the surrounding tubules; *D*, disintegration of the softened dentin matrix caused by the action of saprophytic microorganisms.



FIG. 118.—Advanced caries of the dentin. Formation of clefts at right angles to the dentinal tubules. *ID*, infected dentinal tubules; *C*, clefts in the dentin containing necrotic masses. At *X*, the necrotic content of the clefts has fallen out.

at right angles to the Tomes' fibers, namely, parallel to the dentin surface. In decalcification and subsequent shrinkage of the matrix, these layers of fibrils form natural lines of cleavage along which clefts may form by retraction of the walls. These clefts follow the general direction of the fibrils, at right angles to the dentinal tubules. If there is a considerable amount of decayed dentin, such as in extensive occlusal decay of a molar, large clefts form in the decalcified dentin parallel to the original dentin surface, while the general progress of caries occurs at right angles to the surface (Fig. 119). This arrangement of the clefts in the softened dentin facili-

tates the removal of the decay: the carious dentin peels off in layers or flakes, separating easily from the underlying hard dentin.

Finally, caries of secondary dentin will be considered briefly. Secondary dentin formation and its significance as a protective

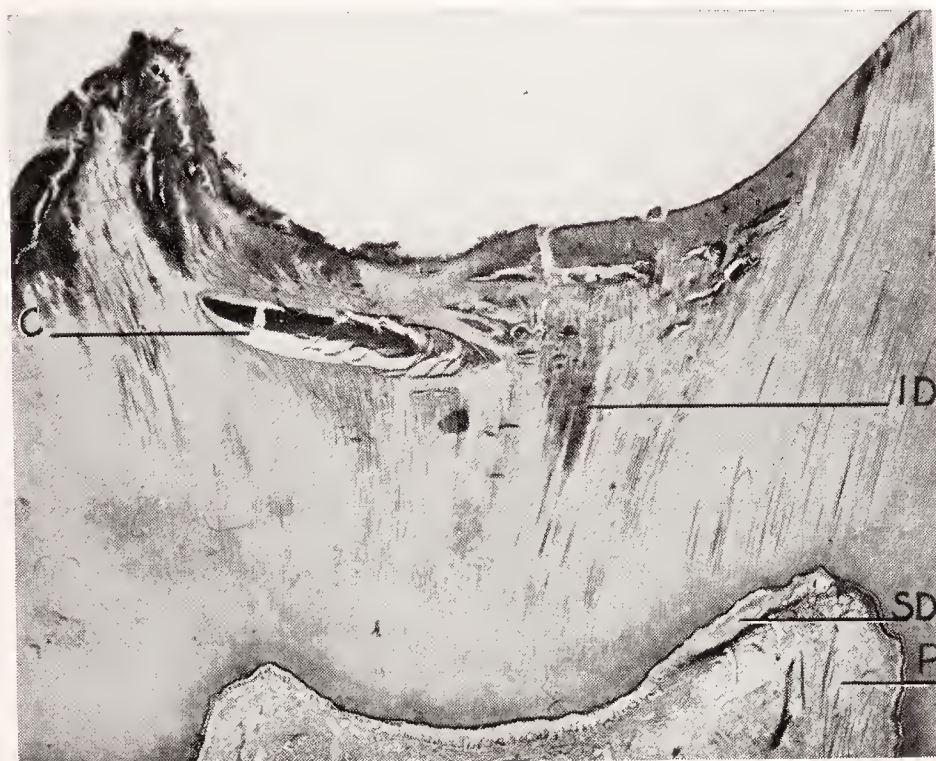


FIG. 119.—Extensive occlusal caries in a lower molar. Formation of clefts in the carious dentin. *C*, large cleft parallel to the dentin surface; *ID*, infected dentinal tubules; *SD*, secondary dentin; *P*, pulp.

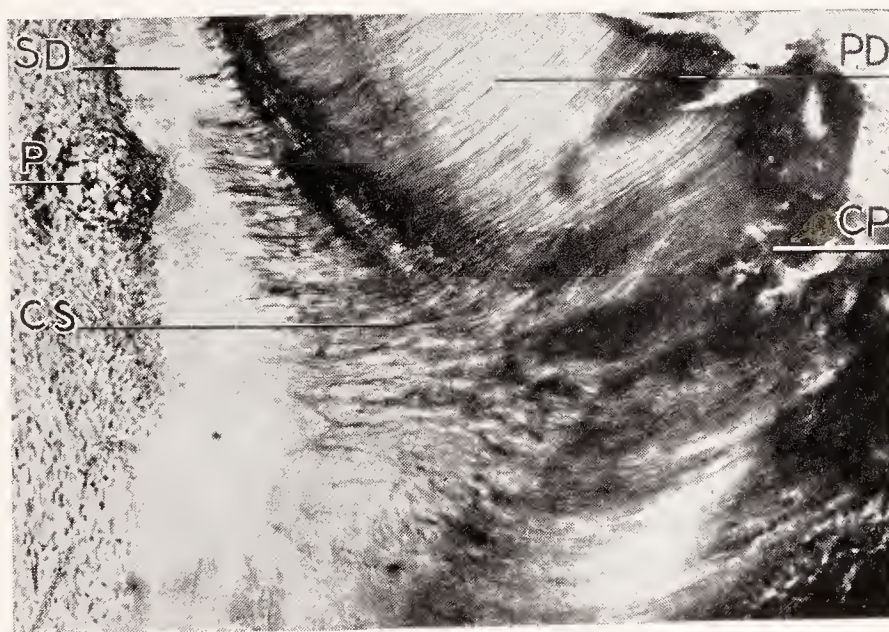


FIG. 120.—Caries of secondary dentin. *PD*, primary dentin; *CP*, caries of the primary dentin; *SD*, secondary dentin; *CS*, caries of secondary dentin; *P*, pulp in a state of incipient inflammation (pulpitis).

measure against caries were illustrated in the preceding chapter. Sooner or later caries progressing through the primary dentin reaches the border between primary and secondary dentin and involves the latter (Fig. 120). The progress of caries in secondary dentin is usually somewhat slower because of the smaller number of den-

tinal tubules. The microscopic appearance of decayed secondary dentin is characterized by the irregular distribution and invasion of microorganisms caused by the irregularity of the tubules (Fig. 121).

In older individuals, in whom part of the root is exposed, plaques may form on the root surface, and caries of the cementum may

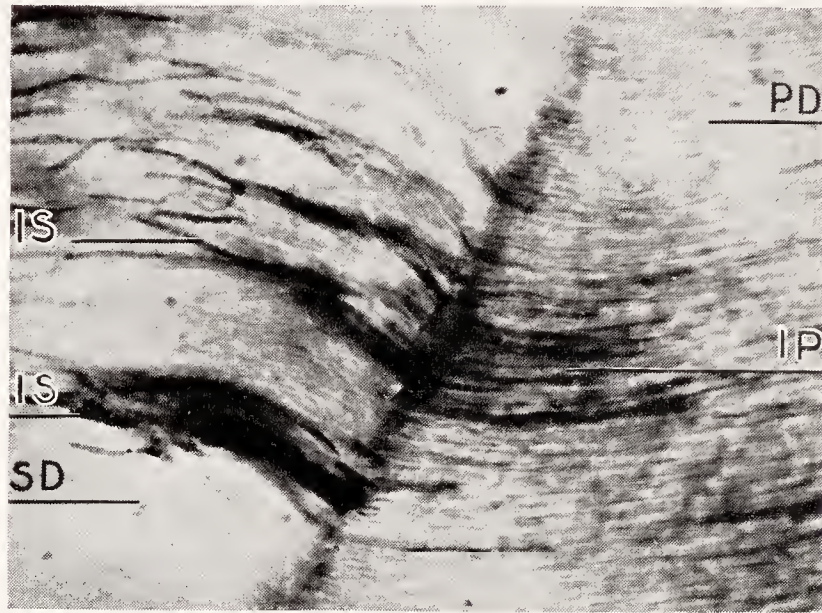


FIG. 121.—Caries at the borderline between primary and secondary dentin. *PD*, primary dentin; *IP*, infected tubules of the primary dentin; *SD*, secondary dentin; *IS*, infected irregular tubules of the secondary dentin.



FIG. 122.—Caries of the cementum. *PC*, primary cementum covered by a plaque; *CC*, caries and disintegration of the cementum; *CD*, caries of the dentin.

develop. The microorganisms follow the course of the former Sharpey's fibers; soon the cementum is decalcified and the underlying dentin is involved (Fig. 122).

Chronic and Arrested Caries.—Chronic caries of the dentin is occasionally encountered on the occlusal surfaces of teeth. The exposed dentin is yellow, brown, or black; it has a polished appearance, and is hard. This condition is known as eburnation (hardening). The cause for this is that in the course of extensive occlusal

caries, the surrounding enamel walls break off. The softened dentin is exposed and worn away by mastication until the hard, intact dentin at the bottom of the cavity is reached. The smooth surface and the impossibility of the lodgment of fermenting substances and microorganisms prevent further decay. Under the microscope a limited number of microorganisms is found in the superficial portion of the exposed dentinal tubules; however, the lack of food retention and the absence of plaque formation on the surface inhibit further development of these microorganisms. The deeper layers of dentin are extensively sclerosed; the pulp chamber is usually greatly reduced in size by secondary dentin formation. Exposure of the pulp is avoided by the extensive deposition of secondary dentin under the decayed portion of the crown.

Occasionally reference is made in dental literature to "healed caries," to indicate that no further destruction or dissolution of the dental tissues occurs in a carious lesion. In the author's opinion the use of the word "healed" is not justified. There is no evidence that carious dentin, once it has been decalcified, ever recalcifies or hardens. Therefore, the term "arrested caries" should be used to describe the condition in which decay has come to a standstill. A good example of arrested caries is the brown marks found on the approximal surfaces of teeth after decayed adjacent teeth have been lost. These brown spots, which otherwise would probably have developed into typical approximal caries, may remain unchanged for decades after the tooth surface has been made a self-cleansing area. The impossibility of food retention and plaque formation prevents further progress of the bacteria in the enamel. The observation of arrested caries as a result of a changed dietary régime can be explained in a similar manner. The progress of the decalcifying microorganisms is arrested, or at least greatly retarded; the softened dentin is lost or worn away, so that finally the discolored, sound, hard dentin at the bottom of the cavity is exposed.

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CHAPTER VI.

PULPITIS.

THE pulp is a sensitive organ which reacts readily to any irritation or injury. Injury to the pulp usually causes inflammation, known as pulpitis, but a severe injury may kill the pulp.

Minor injuries, such as abrasion or superficial caries, do not cause an inflammatory reaction of the pulp tissue but merely stimulate the deposition of secondary dentin. An inflammatory reaction is the result of more severe injuries that expose or nearly expose the pulp. In the latter case, when there is only a thin layer of dentin between the irritant and the pulp, the individual resistance of the pulp tissue plays an important rôle. Sometimes deep caries or a large filling causes the formation of secondary dentin, and the pulp tissue does not become inflamed; in another tooth a similar cavity or filling may cause pulpitis. In every tissue reaction the resistance of the cells is of decisive influence, and since the limit of this resistance is unknown, it is impossible to determine how much irritation is necessary to produce a certain form of tissue reaction.

The microscopic manifestations of pulpitis are the same as those of inflammation of other tissues; however, there is a basic difference between the final outcome of pulpitis and that of inflammatory processes elsewhere in the body. Nearly all tissues can survive a certain amount of inflammation, which may terminate by resolution or by scar formation, but the organ as a whole survives and continues to function. This is not true, however, of the pulp. Clinical experience demonstrates that sooner or later inflammation of the pulp tissue causes its complete destruction. If the injury is slight and the pulp resistant, the inflammatory process may persist for years, and there may be a marked tendency of the tissue to wall off the progressing destruction. The ultimate outcome, however, is always necrosis of the entire pulp.

The unfavorable outcome of pulpitis is largely due to the location of the pulp within a rigid hard-tissue capsule. The defensive mechanisms of the vascular system of the pulp are limited by the absence of a collateral circulation. The typical manifestations of inflammation, hyperemia, edema, and cellular infiltration, can occur only as far as the surrounding unyielding walls will permit. Moreover,

any edema of one portion of the pulp will cause disturbed circulation in the rest of the pulp tissue. Another basic difference between an exposure of the pulp and a wound elsewhere in the oral cavity is that there is no epithelial tissue in the pulp to play a part in the reparative process. Every exposure of connective tissue of the oral cavity heals by epithelization. In a pulp wound no such healing is possible.

Pulp Capping.—The only way in which an exposed pulp can heal is by deposition of dentin in the injured area, which would form a solid cover over the exposure. This form of pulp healing is possible, and the different methods of pulp capping are based upon this possibility. However, pulp exposure and pulp capping in adults are often followed by inflammation or necrosis of the pulp tissue. Therefore, such attempts should, as a rule, be discouraged, with the exception of children's teeth whose wide foramina provide a better blood supply and in which the high tissue resistance offers a greater chance for success.

The recent work of Hess, Neuwirt, and Müller has produced evidence that a healthy young human pulp may survive accidental exposure. In the absence of infection the pulp tissue produces secondary dentin which seals the area of exposure. Various drugs have been used to cover the exposed pulp tissue; of these zinc oxide-eugenol and calcium preparations appear the most promising.

The entire problem of pulp capping and the saving of exposed, healthy pulps is still in the experimental stage. The greatest difficulty is the fact that there is no way of determining whether or not an exposed pulp is already invaded by bacteria and inflamed. Once pulpitis has started, any attempt to cap the pulp is bound to fail.

THE ETIOLOGY OF PULPITIS.

According to etiology, three forms of pulpitis can be distinguished:

Pulpitis due to bacterial invasion.

Pulpitis due to chemical irritation.

Pulpitis due to thermal irritation.

The first form, infective pulpitis, is the most frequently encountered. It is caused by an invasion of the pulp tissue by bacteria from a carious cavity or from accidental pulp exposure in tooth fracture or cavity preparation.

Chemical pulpitis is caused by the action of drugs, such as arsenic trioxide. Silicate cements are sometimes responsible for chemical pulpitis and death of the pulp; it is not an uncommon observation

that a pulp becomes inflamed and dies under a silicate filling, probably as the result of diffusion of some cell poison from the filling material through the dentin into the pulp.

The third form, pulpitis due to thermal irritation, occurs under large deep-seated metal fillings without proper insulation. After the insertion of such fillings thermal stimuli, especially cold, are transmitted to the pulp and cause pain. The outcome depends largely upon the individual resistance of the pulp. Some pulps are able to protect themselves by secondary dentin; in others, pulpitis develops as the result of thermal irritation.

Hematogenous Pulpitis.—On rare occasions pulpitis occurs in intact teeth. Since the only way in which bacteria can enter the pulp of an intact tooth is by way of the blood stream, such a pulpitis is called hematogenous. It is observed in patients with septicemia, in whose blood pathogenic microorganisms are circulating. Lieck reported several cases of pulp abscesses in the otherwise intact teeth of patients who had died of septic infections. In the course of the disease the bacteria had been disseminated by the blood stream, and among other tissues had reached the pulps of the teeth and there caused an inflammatory reaction.

HYPEREMIA OF THE PULP.

Pulpitis is frequently preceded by a state of hyperemia of the pulp. Clinically, pulp hyperemia is characterized by marked sensitiveness to thermal changes, especially to cold. This sensitiveness is of temporary character; it occurs usually as the result of stimulation by cold water or cold food and subsides as soon as the irritation is removed. The affected tooth usually has a deep cavity or a deep-seated metal filling. Microscopically, dilatation and engorgement of the pulp vessels are found; the outline of the vessels is slightly irregular, indicating the beginning of injury to the vessel walls (Fig. 123).

Hyperemia of the pulp is a reversible condition. If the irritation is removed, the pulp may return to normal; the blood-vessels contract, and normal circulation is reestablished. Sometimes, however, there is a gradual transition from hyperemia to pulpitis, and then the pulp is lost.

In clinical practice the diagnosis of hyperemia is often made when there is merely increased sensitiveness of exposed dentin. In many cases of so-called hyperemia of the pulp under a deep cavity, the dentin itself is extremely sensitive to touch; this suggests hypersensitive dentin rather than an actual change in the pulp tissue.

ACUTE PULPITIS.

In pulpitis, as in every inflammatory process, an acute and a chronic form can be distinguished. Clinically, the acute form usually causes pain, whereas chronic pulpitis causes little or no pain. Microscopically, acute pulpitis shows all the characteristics of an acute inflammation: enlargement of the blood-vessels, slowing of the circulation, accumulation of leukocytes in the periphery of the blood stream, migration of these cells through the vessel wall, diffu-

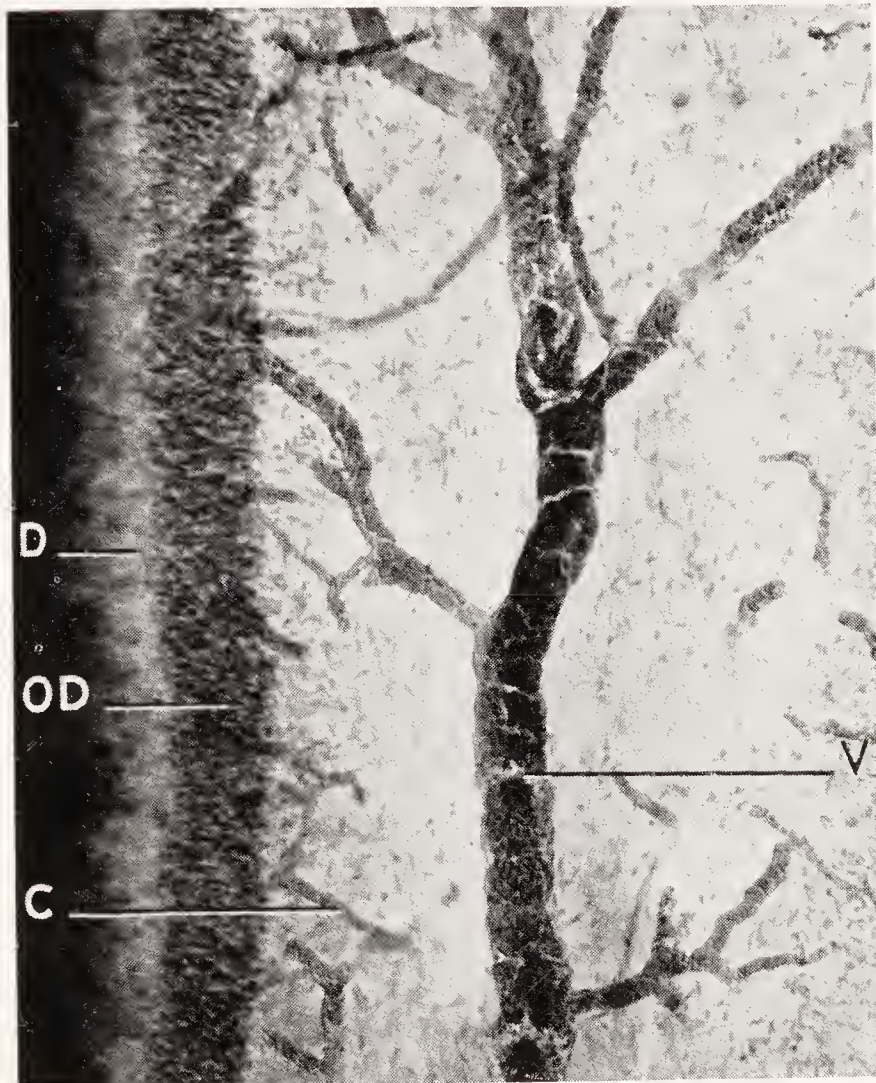


FIG. 123.—Hyperemia of the pulp. Dilatation and hyperemia of the pulp vessels. The pulp tissue between the vessels is free from inflammatory changes. *V*, hyperemic blood-vessel (vein); *C*, capillaries near the odontoblastic layer; *OD*, odontoblasts; *D*, predentin.

sion of blood serum through the walls of the vessels, and mobilization of the cells of defense in the pulp tissue. Subsequently the pulp tissue breaks down and a pulp abscess develops. In chronic pulpitis reparative processes can be observed in addition to the destructive changes. A chronic ulcer or granulation tissue is formed on the surface of the inflamed pulp, which may persist for years without clinical symptoms.

Depending upon the symptoms and the amount of pulp tissue involved, acute pulpitis can be divided into acute partial and acute

total pulpitis. However, the clinical symptoms do not always correspond to the pathohistological findings. Sometimes a pulp that has caused intense pain shows microscopically only a small area of inflammation; again, pulps that microscopically show extensive suppurative pulpitis may have caused only slight, indefinite pain. Variations in the subjective reaction to pain, differences in the location of the pulpitic area, and the presence or absence of pulp nerves

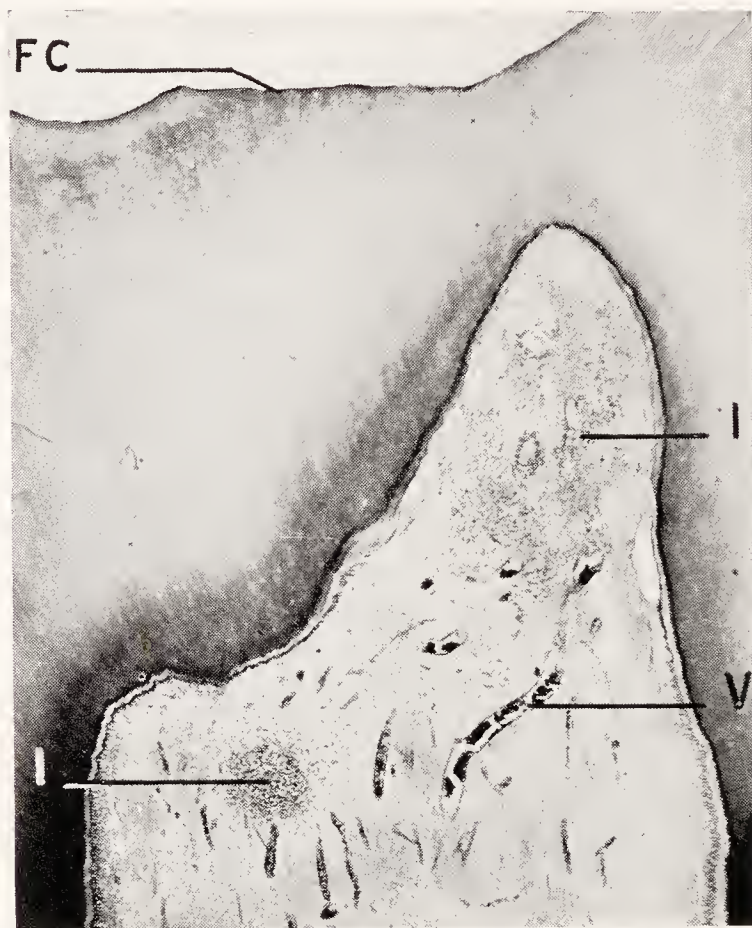


FIG. 124.—Acute partial pulpitis (serous form) under a filling. Upper molar. *FC*, floor of cavity; *I*, areas of cellular infiltration of the pulp tissue; *V*, hyperemic blood-vessels.

in the diseased portion of the pulp are some of the factors responsible for the occasional discrepancies between clinical history and microscopic findings.

Acute Partial Pulpitis.—Acute partial pulpitis is diagnosed clinically by pain in a tooth with a large cavity or filling. The attacks of pain are elicited by changes in temperature, especially by cold fluids or cold air. After the thermal irritation has subsided, the pain continues for a while. The pain also occurs spontaneously, particularly at night. Usually the involved tooth has extensive caries extending into a pulp horn. The tooth is not sensitive to percussion. Microscopically, a serous and a purulent form of acute partial pulpitis can be distinguished.

The serous form of acute partial pulpitis represents the earliest stage of pulpitis, developing from a hyperemia of the pulp by the passage of white blood cells and the diffusion of blood serum through

the walls of the pulp capillaries (Fig. 124). Hyperemia of the pulp vessels in the vicinity of the cavity, the presence of polymorphonuclear leukocytes in the pulp tissue, and edema of the pulp tissue are the microscopic characteristics of the earliest form of acute pulpitis. Of the pulp cells the odontoblasts are most vulnerable; they are usually missing in the inflamed area. In Figure 125 a microscopic area of pulpitis is illustrated. Inflammatory round cells are massed around a capillary in the pulp tissue. All capillaries in the neighborhood are dilated and hyperemic; the surrounding connective tissue of the pulp is edematous.

The serous form of pulpitis gradually changes into purulent pulpitis. As a result of the action of bacteria and leukocytes, the pulp

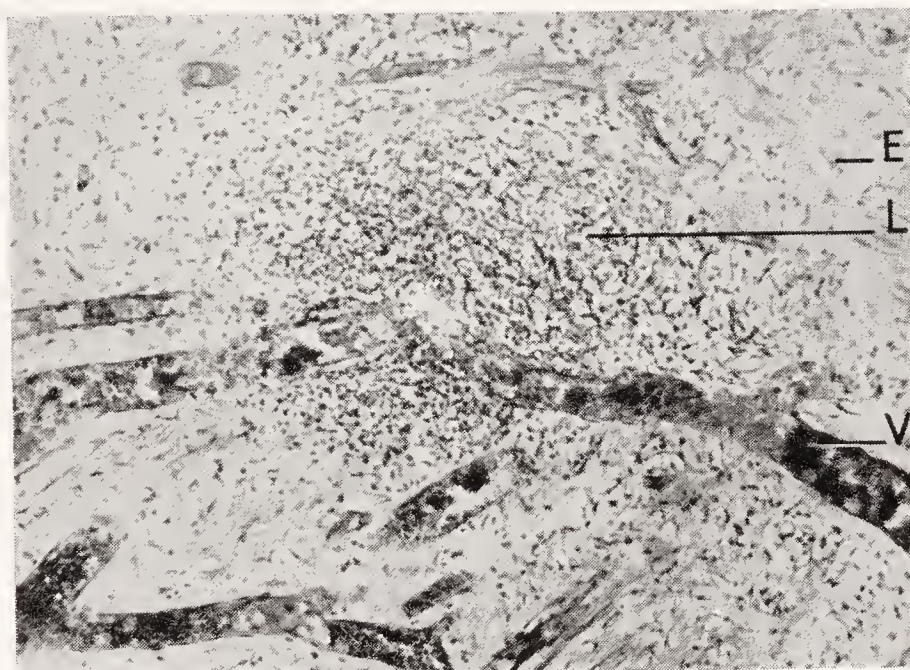


FIG. 125.—Higher magnification of Fig. 124. Accumulation of inflammatory exudate cells around the capillaries of the pulp. *L*, exudate cells (leukocytes); *V*, hyperemic blood-vessels; *E*, edema of the pulp tissue.

tissue breaks down; small cavities containing pus develop in the pulp. These are known as pulp abscesses.

Figure 126 shows a lower bicuspid with an early stage of a pulp abscess in acute partial pulpitis. Because of caries, secondary dentin was deposited on the inner wall of the pulp chamber, but it offered insufficient protection, and the bacterial invasion reached the pulp horn. The latter is densely infiltrated with leukocytes; the pulp tissue adjacent to the carious dentin broke down and was replaced by a small abscess cavity containing leukocytes and cell debris. The blood-vessels in the immediate neighborhood of the pulpitic area are hyperemic. Beyond this area the pulp tissue is normal.

A slightly advanced stage of partial pulpitis with formation of an abscess is seen in Figure 127; the conditions are similar to those shown in the preceding illustration. The incisal portion of the pulp



FIG. 126.—Acute partial pulpitis (purulent form) under penetrating caries. Lower bicuspid. *C*, carious destruction reaching the pulp horn; *SD*, secondary dentin; *I*, dense inflammatory infiltration of the pulp tissue with beginning abscess formation; *P*, normal pulp tissue with slightly dilated blood-vessels.

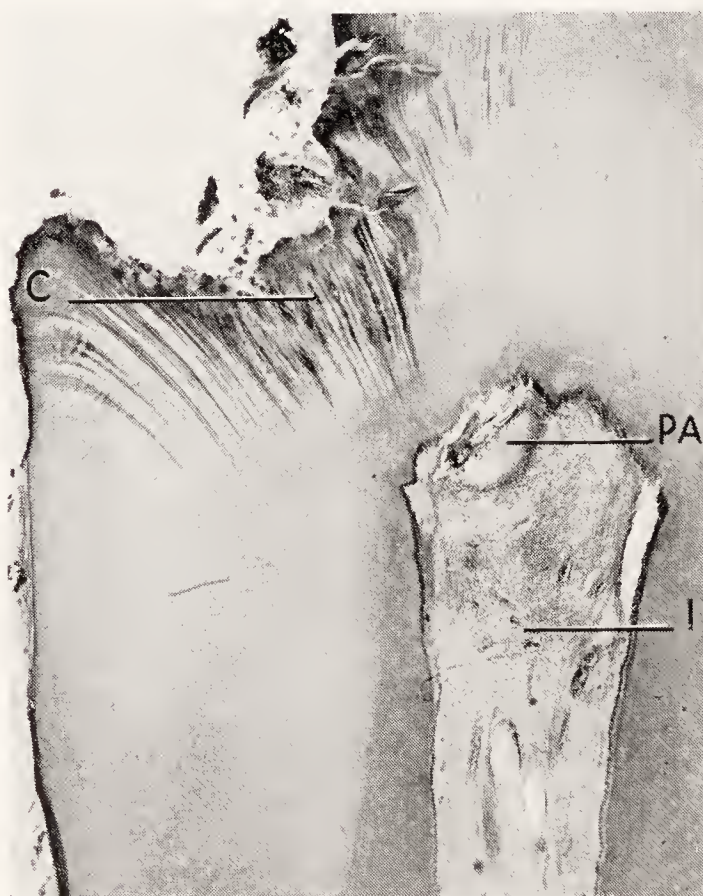


FIG. 127.—Acute partial pulpitis under deep caries. Formation of a pulp abscess. Lower bicuspid. *C*, caries of the dentin; *PA*, pulp abscess; *I*, infiltrated pulp tissue.

is densely infiltrated. Near the carious cavity a small abscess cavity has developed in the pulp tissue in which, under higher magnification, a large number of polymorphonuclear leukocytes are visible

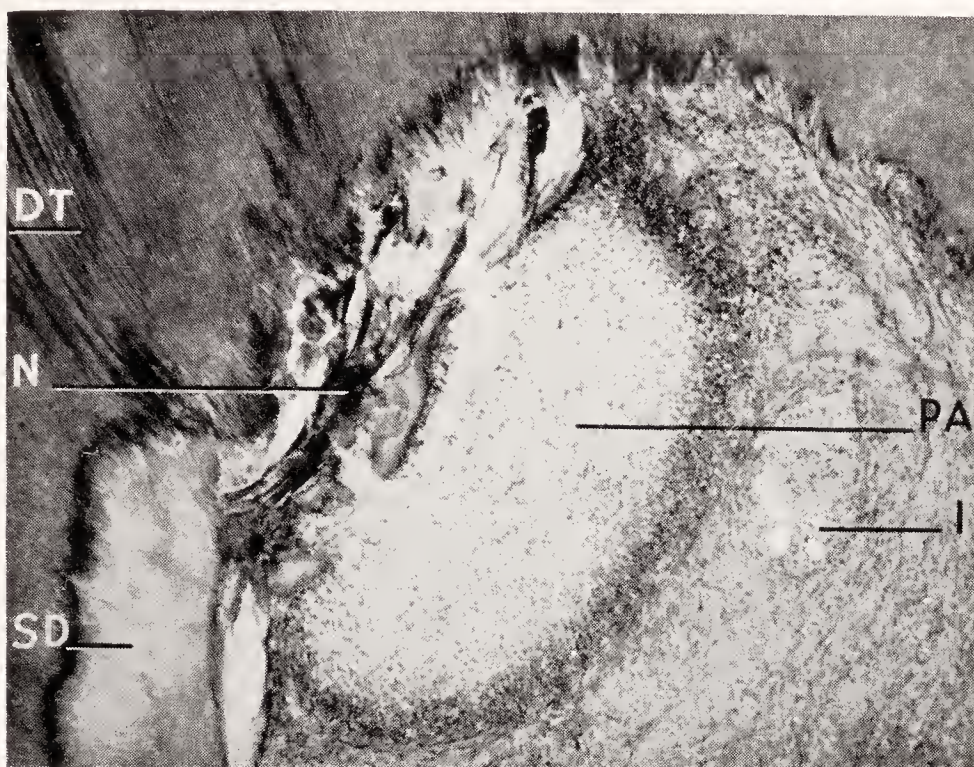


FIG. 128.—Higher magnification of Fig. 127. Pulp abscess. *DT*, infected dentinal tubules; *N*, necrosis of dentin and pulp tissue; *PA*, pulp abscess, the result of the breaking down of the pulp tissue next to the infected dentin; *I*, densely infiltrated pulp tissue; *SD*, secondary dentin.

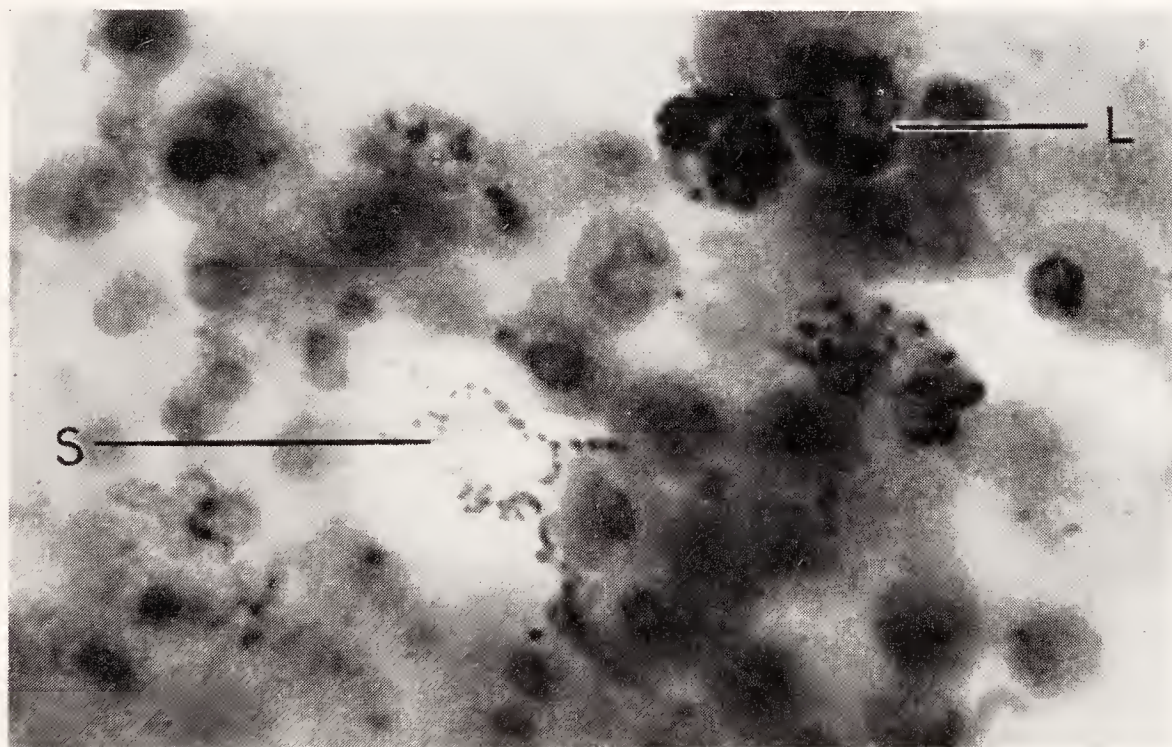


FIG. 129.—Cocci in the purulent exudate from the surface of an inflamed pulp. *S*, streptococci lying between the exudate cells; *L*, leukocytes containing cocci (phagocytosis).

(Fig. 128). In Gram-stained specimens of the pus from purulent pulpitis, numerous pathogenic microorganisms can be seen (Fig. 129). They are arranged in chains (streptococci); some of them are located

between the white blood cells, while others are engulfed by leukocytes and monocytes and are inside the cytoplasm of these cells.

The pulp tissue nearer to the root is normal except for a slight enlargement of the blood-vessels. In some of the vessels an increased number of polymorphonuclear leukocytes can be observed; the surrounding pulp tissue, however, is normal.

Acute pulpitis, like any other acute purulent inflammation, is microscopically characterized by the predominance of neutrophil, polymorphonuclear leukocytes in the cellular exudate.

Bacteriology of Pulpitis.—Knowledge of the distribution of pathogenic bacteria in a diseased pulp is helpful in understanding the pathology of pulpitis. For a study of this kind, sections through pulpitic teeth are stained with Gram stain and examined for the presence of microorganisms in the various levels of pulp and dentin. In addition, cultural methods may be used for the identification of these bacteria.

In hyperemia of the pulp no bacteria are found in the pulp tissue. There may be some in the dentinal tubules in the vicinity of the pulp, but they have not yet invaded the pulp chamber and, consequently, there is no pulp inflammation.

In the early stages of pulpitis, when there is merely a serous and cellular infiltration of a pulp horn, the pulp tissue may still be free of microorganisms. Invariably, however, there are masses of bacteria, usually cocci, in the tubules of the decayed dentin near the pulp; it has been suggested that toxins produced by these bacteria penetrate to the pulp and are responsible for the inflammatory reaction of the pulp tissue (Harndt).

In partial purulent pulpitis and abscess formation, bacteria are always found in the pulp tissue. They are located in the inflamed tissue and in the purulent exudate (see Fig. 129). However, by no means are they evenly distributed throughout the pulp. The center of the inflamed portion of the pulp, the pulp abscess, is completely surrounded by a layer of bacteria. (Fig. 130.) Farther away from the abscess, the number of bacteria decreases rapidly, and the histologically normal portion of the pulp tissue is free of microorganisms. This shows that the infective process and the tissue reaction in the pulp are fundamentally the same as in a purulent infection elsewhere in the body: bacteria are found in the area of inflammation; in the periphery of the inflamed region, however, the spread of infection is successfully checked by the defense mechanism of the body, mainly by the inflammatory exudate cells, and no bacteria are found in the surrounding tissues.

Only in total pulpitis is there a diffuse permeation of most of the pulp tissue with bacteria. But even then the root canals may be free of infection. In chronic pulpitis (ulcerated pulp, pulp polyp) there is always a mixed infection; the exposed pulp surface teems with all kinds of oral microorganisms, while the deeper layers of inflamed pulp or granulation tissue are usually free of bacteria.

Acute Total Pulpitis (Suppurative Pulpitis).—Acute total pulpitis is a condition in which the major portion of the pulp tissue is involved in an acute inflammation. It develops from acute partial pulpitis

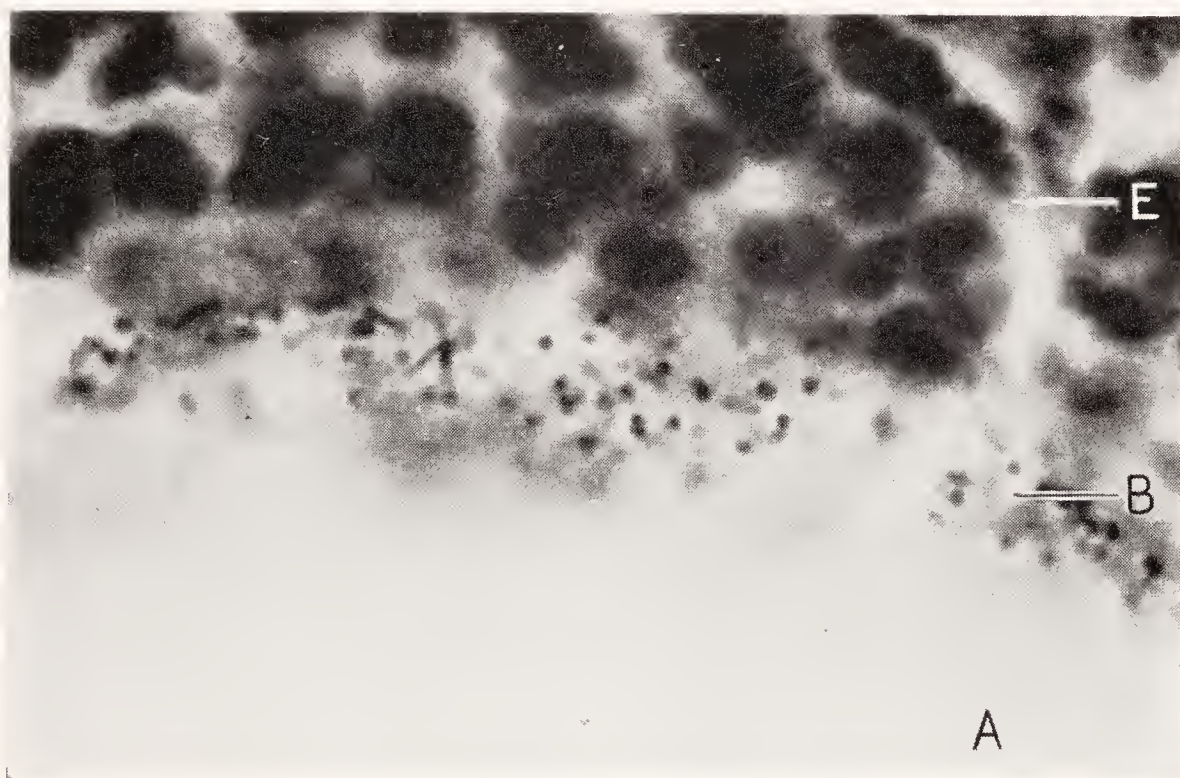


FIG. 130.—Bacteria in the wall of a pulp abscess. *A*, abscess cavity; *B*, bacteria (rods and cocci); *E*, exudate cells.

by the spreading of the inflammation over the entire pulp. There is no sharp borderline between partial and total pulpitis, and many intermediate stages are encountered both microscopically and clinically. The transition from partial pulpitis with occasional attacks of pain to total pulpitis with continuous throbbing pain is a gradual one, usually developing over a period of several days. In a typical case of total pulpitis the pain is severe; the patient is kept awake at night. Thermal changes increase the pain, although the pulp is usually more sensitive to heat than to cold. The tooth may be slightly sensitive to percussion, indicating an incipient involvement of the apical periodontal membrane.

In the microscopic examination of extracted teeth with the clinical diagnosis of acute total pulpitis, it is usually found that those teeth in which the inflamed pulp is still covered by a layer of carious dentin or by a filling have caused the greatest pain because of the

pressure that develops if the pus is confined within the pulp cavity. Those teeth with total pulpitis in which the purulent exudate can drain into the oral cavity usually cause less severe pain, since no pressure is exerted upon the nerves of the inflamed pulp.

The final outcome of total pulpitis seems to depend largely upon this difference between the open and closed forms. In closed pulps from which the exudate has no drainage, infiltration of the entire pulp tissue, abscess formation, and necrosis of the pulp occur rather quickly. In teeth with pulps in communication with the oral cavity, the transition to a chronic form of pulpitis is frequently observed.

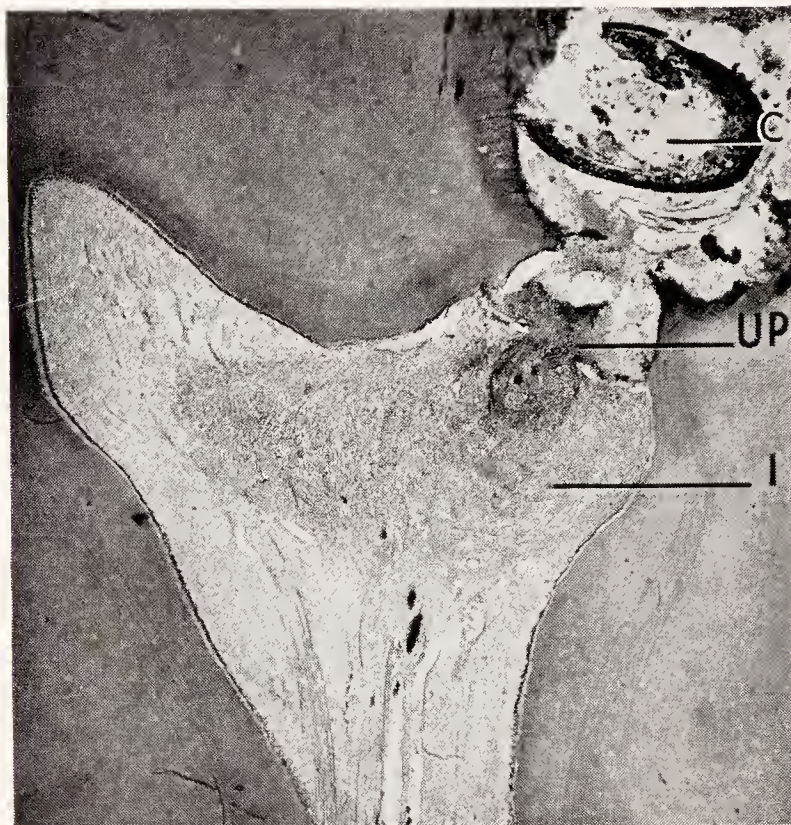


FIG. 131.—Acute total pulpitis (open form). Upper molar. *C*, carious cavity containing food debris; *UP*, ulcerated exposed pulp surface at the bottom of the cavity; *I*, diffuse inflammatory cell infiltration in the crown portion of the pulp.

The drainage of exudate prevents pressure and allows the pulp tissue to establish a temporary balance between advancing infection and defensive reaction of the pulp. This condition will be described more in detail under the heading of chronic pulpitis.

Acute Total Pulpitis, Open Form.—In the open form of acute total pulpitis, a large portion of the pulp or the entire pulp is in a state of acute, purulent inflammation; the communication with the oral cavity is usually through a carious cavity. In Figure 131, in the upper right corner of the picture, can be seen a carious cavity, which is filled with debris, broken-down dentin, pus cells, and food particles. One of the pulp horns is exposed, and from its surface a purulent exudate is being discharged. Part of the pulp tissue has been

destroyed, and most of it is densely infiltrated with inflammatory exudate cells. The blood-vessels are dilated and hyperemic; the odontoblastic layer has completely disappeared near the area of pulp exposure and shows degeneration in the more distant parts of the pulp surface. As a whole, this condition differs from acute partial pulpitis (Figs. 126 and 127) only in the degree and the extent of inflammation, for, instead of only one small area of pulp tissue, the entire crown portion of the pulp is involved.

If caries and pulp exposure are found in the cervical portion of a tooth, pulpitis starts in the root canal next to the area of exposure and from there spreads crownward into the pulp chamber and rootward into the canal. Such a condition is seen in Figure 132, illus-



FIG. 132.—Acute total pulpitis (open form) under penetrating gingival caries. Lower molar. *C*, carious cavity on the distal surface of the distal root; *PA*, pulp abscess in the distal root canal extending upward into the crown portion of the pulp; *I*, diffuse inflammatory infiltration of the crown portion of the pulp.

trating a specimen of a lower molar with clinical symptoms of total pulpitis. The occlusal portion of the crown is intact, but there is deep caries on the distal surface of the crown, extending downward into the root canal. The microscopic examination shows that most of the distal root canal is filled with pus and that an abscess cavity has developed in the pulp tissue crownward from the carious cavity. The entire pulp is in a state of diffuse, purulent inflammation, the intensity of which decreases toward the mesial pulp horn.

Acute Total Pulpitis, Closed Form.—In the closed form of total pulpitis the formation of abscess cavities in the pulp tissue is the typical microscopic finding. The purulent exudate, which cannot

escape, accumulates under pressure inside the pulp chamber and causes large areas of tissue destruction. When such a pulp abscess is exposed by removing the overlying dentin, a drop of pus emerges from the opening, seemingly under pressure. This is usually followed by relief from the pain, comparable to the relief that follows lancing an abscess, evacuating the pus, and removing the pressure of the retained exudate.

Suppurative pulpitis in a closed pulp chamber usually causes rapid breaking-down and necrosis of the entire pulp. Such a condition is shown in Figure 133. In a lower molar total pulpitis had developed

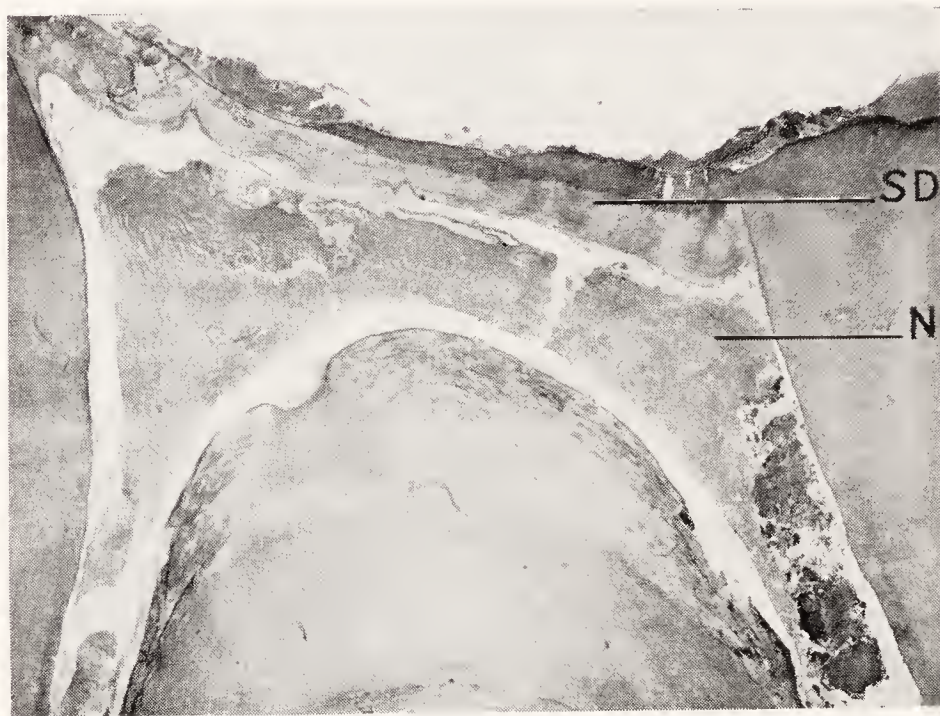


FIG. 133.—Necrosis of the pulp. Lower molar. *SD*, layer of secondary dentin covering the pulp chamber; *N*, necrotic tissue in the pulp chamber and in the root canals.

under extensive caries that almost reached the pulp; only a thin, still intact layer of dentin covers the pulp chamber. Clinically and microscopically the pulp shows evidence of necrosis. The cells have lost their staining properties, but pulp calcifications and dead exudate cells can still be recognized in the tissue remnants. Farther apically in the root canals, there is some living inflamed pulp tissue still present.

CHRONIC PULPITIS.

The outcome of total pulpitis may be twofold: either rapid destruction of the entire pulp or transition into chronic pulpitis. The latter outcome is encountered mostly in young individuals with high tissue resistance, in infections of low virulence, and in those teeth in which drainage of the inflammatory exudate is possible.

Two forms of chronic pulpitis can be distinguished clinically and microscopically: an ulcerative and a hyperplastic form.

Chronic Ulcerative Pulpitis.—In chronic ulcerative pulpitis an ulcer develops on the surface of the pulp, while in the underlying pulp tissue there is a dense zone of round cells. This condition, which causes few, if any, clinical symptoms, may persist for a long time. Upon clinical examination, a cavity leading into the pulp is usually found; the tooth shows little or no reaction to cold or heat. Living, bleeding pulp tissue is still present, which is much less sensitive to touch than a normal or acutely inflamed pulp, probably because of degeneration of the nerves in the pulp stump.

Microscopically, there is a tendency toward incapsulation of the inflammatory process and formation of granulation tissue in the

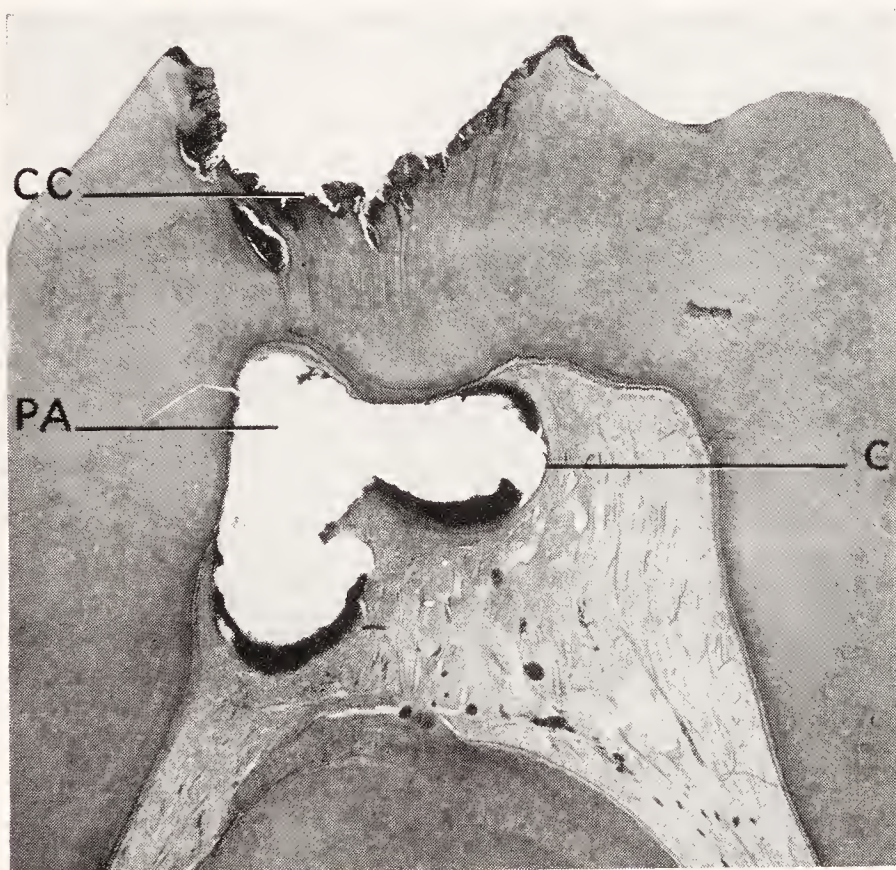


FIG. 134.—Pulp abscess under occlusal caries in an upper molar. The abscess is surrounded by a connective tissue capsule. The rest of the pulp tissue shows only slight inflammatory changes. *CC*, carious cavity; *PA*, pulp abscess; *C*, capsule of pulp tissue surrounding the abscess cavity.

pulp. The transition from acute suppurative pulpitis into chronic pulpitis is illustrated in Figure 134, which shows an abscess cavity in the pulp of a lower first molar under occlusal caries. The rest of the pulp tissue has been altered very little; the odontoblastic layer on the distal wall and on the floor of the pulp chamber is microscopically intact. The pulp tissue is arranged parallel to the wall of the abscess cavity, thus forming a fibrous capsule around the abscess. This condition indicates considerable resistance of the pulp tissue: an attempt was made by the pulp to wall off the spread of inflammation and to incapsulate the abscessed area.

Another form of chronic ulcerative pulpitis is seen in Figure 135. The entire pulp of a lower second deciduous molar is in a state of chronic inflammation and contains two abscess cavities. Caries has destroyed a part of the pulp tissue in the mesial pulp horn; the ulcerated pulp surface is covered by an irregular calcified mass that

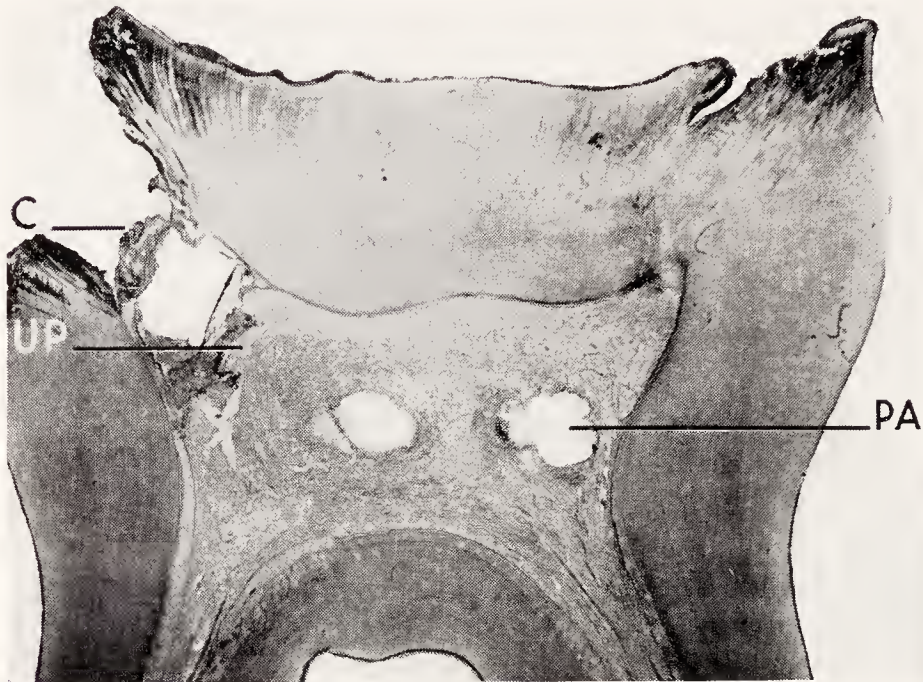


FIG. 135.—Chronic ulcerative pulpitis. Lower deciduous molar. All of the pulp tissue shows extensive diffuse round-cell infiltration. *C*, carious cavity; *UP*, ulcerated surface of the pulp; *PA*, abscess cavities within the pulp tissue.



FIG. 136.—Higher magnification of Figure 135. *P*, densely infiltrated pulp tissue; *CP*, irregular calcification of the pulp tissue covering the area of pulp exposure; *PD*, primary dentin.

extends across the opening, leaving only a small communication between pulp and cavity (Fig. 136). Such calcific deposits are frequently found in chronically inflamed pulps; they develop by a calcification of the dying or necrotic tissue and may be considered

an attempt to heal the exposed pulp horn. The presence of such calcifications always indicates that the pulp inflammation is of long standing.

Sometimes, although the crown portion of the pulp is completely destroyed, the apical part still contains living tissue in a state of chronic inflammation. On the surface of the pulp stump in the root canal, an ulcer discharges exudate and cells into the empty part of the root canal (Fig. 137). Clinically, this condition is frequently

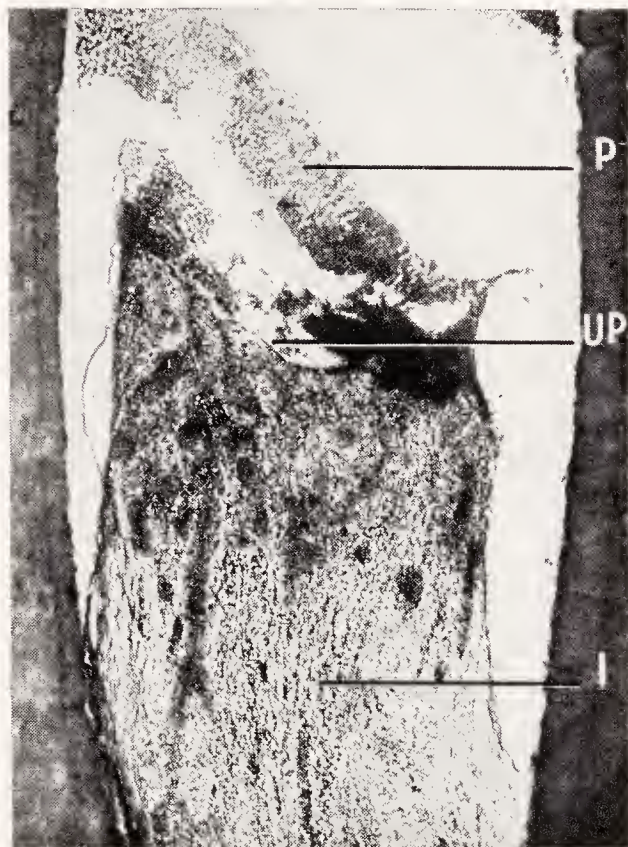


FIG. 137.—Chronic ulcerative pulpitis. Pulp stump in the root canal of a molar after breaking-down of the crown portion of the pulp. *I*, inflamed pulp tissue; *UP*, ulcerated surface of pulp stump; *P*, purulent exudate in the root canal.

found in teeth with deep caries and an exposed pulp chamber containing necrotic pulp tissue; there is no living tissue in the coronal portion of the root canal, but in the apical part the explorer or broach still meets vital, sensitive, bleeding pulp tissue. Coolidge described the histological findings in a tooth of this kind. He called attention to the fact that although the root canals still contained vital, inflamed pulp remnants, the apical periodontal tissues had already undergone marked changes. Enlarged capillaries and an accumulation of plasma cells were found in the periodontal membrane. The continuity of the bone plate around the root end had been lost.

Figure 138 shows a lower molar with penetrating mesial caries. The pulp chamber contains necrotic tissue and debris. The apical portions of the root canals still contain vital, inflamed pulp tissue.

The apical fibers of the periodontal membrane have been destroyed, and the periodontal membrane itself has been widened around the root end (Fig. 139). The soft tissue in this area has lost its normal fibrous structure; irregular fiber bundles are present, between which

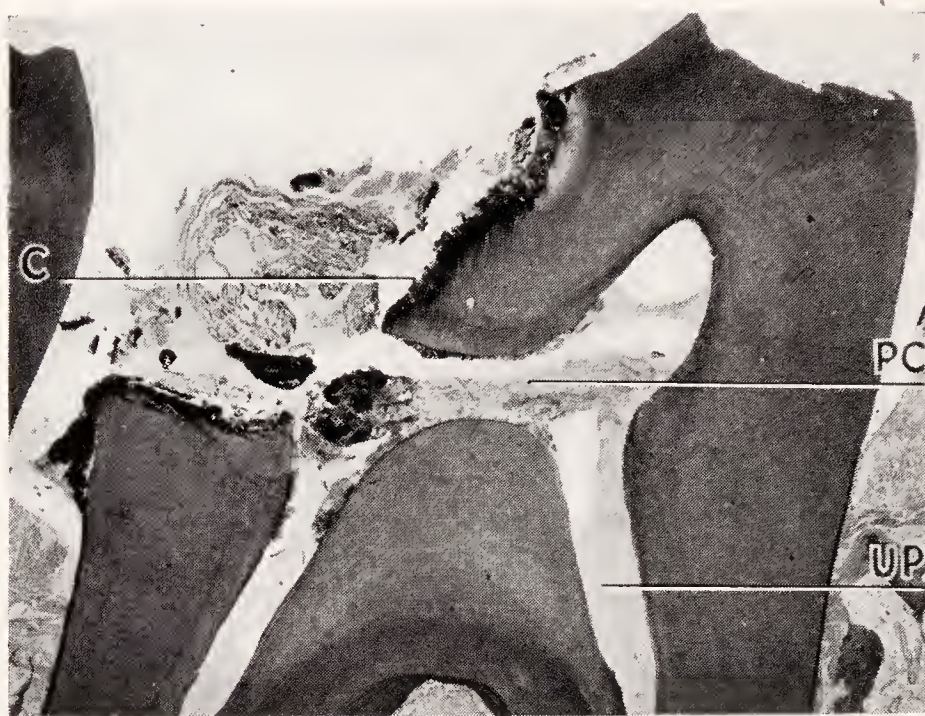


FIG. 138—Chronic ulcerative pulpitis in the root canals of a lower molar. *C*, penetrating caries in the mesial portion of the crown; *PC*, pulp chamber filled with necrotic pulp débris and exudate; *UP*, ulcerated surface of the pulp stump in distal root canal. (Coolidge, Jour. Am. Dent. Assn.)

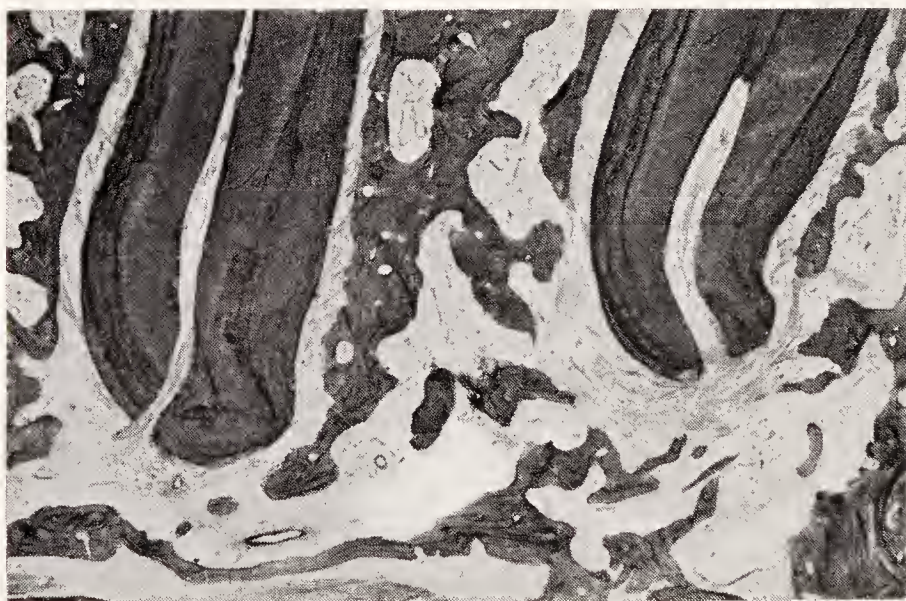


FIG. 139.—Apices of the roots of the molar shown in Figure 138. Both root canals contain vital pulp tissue. There is a break in the inner plate of the alveolar bone at the apex of each root. The apical fibers of the periodontal membrane are destroyed. (Coolidge, Jour. Am. Dent. Assn.)

are wide capillaries and groups of plasma cells (polyblasts). This specimen illustrates the gradual transition from pulpitis to periodontitis. There is no sharp demarcation between these two conditions: although there is still inflamed, vital pulp tissue in the root

canals, the action of the bacteria has already extended beyond the apex. Inflammatory products are probably carried from the ulcer on the surface of the pulp stump through the blood and lymph channels into the periapical tissue and there cause an incipient pericementitis. Radiographically, such a tooth, although it still contains vital pulp tissue, may show a widened apical periodontal space.

Chronic pulpitis in a lower second molar is illustrated in Figure 140. Occlusal decay caused exposure of the distal pulp horn, but instead of the usual progressive destruction of the pulp tissue, the pulp reacted by a generalized inflammation and transformation

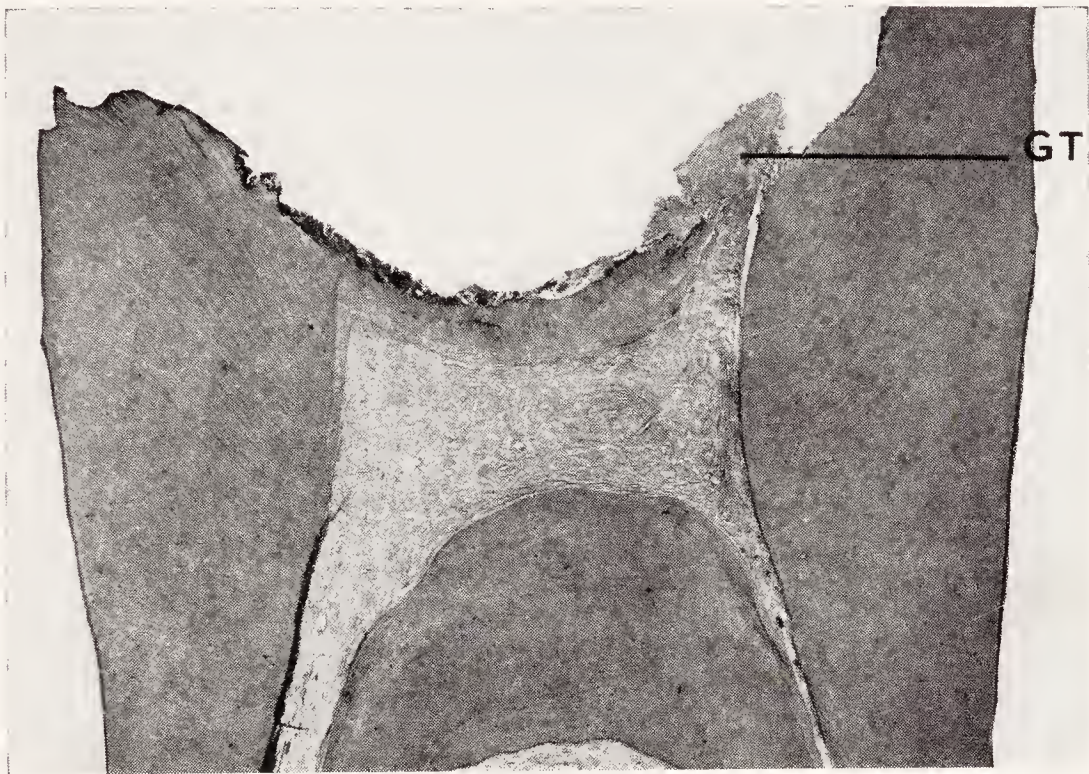


FIG. 140.—Chronic pulpitis following pulp exposure in a lower molar. The pulp has been transformed into granulation tissue. *GT*, plug of granulation tissue proliferating through the opening in the pulp chamber.

of the pulp tissue into granulation tissue. A higher magnification shows nothing of the original pulp structure but, in its place, a new type of tissue, namely, young connective tissue with a large number of inflammatory exudate cells. Some of these are plasma cells, characterized by large, protoplasmic bodies and excentric nuclei; others are small lymphocytes. Both plasma cells and lymphocytes are typical of chronic inflammation. At the point where the pulp is exposed, granulations have proliferated through the small opening in the pulp chamber and appear in the cavity as bright red tissue of pin-head size. This tissue, as well as the rest of the pulp, is only slightly sensitive. No nerve tissue could be found in the entire pulp chamber. The tissue proliferation indicates the transition from the chronic ulcerative type of pulpitis to the chronic hyperplastic form.

Chronic Hyperplastic Pulpitis (Pulp Polyp).—Sometimes the stimulus of a chronic, low-grade inflammation leads to hyperplastic pulpitis. Two factors are essential for the development of this condition: the pulp must be highly resistant, and a communication with the oral cavity must be present. For these two reasons chronic hyperplastic pulpitis is for the most part found in children and adolescents in whom tissue resistance is high, and in broken-down teeth

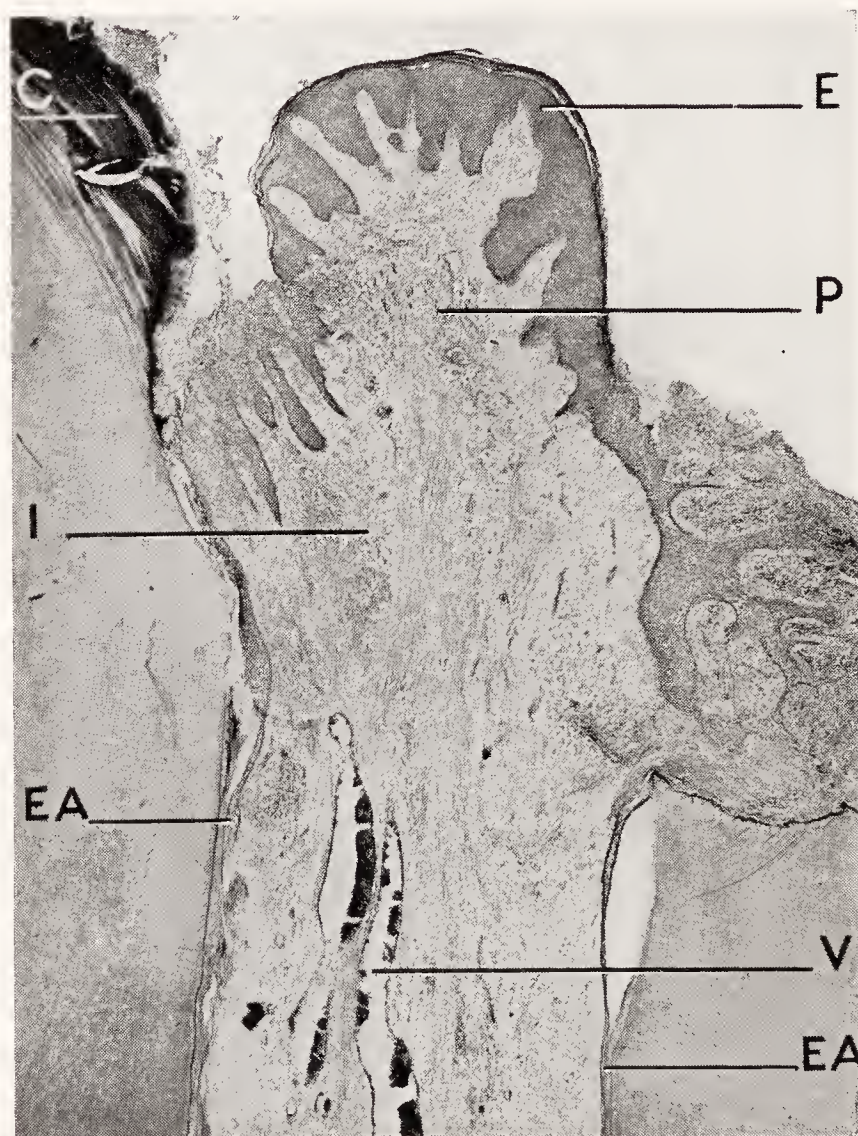


FIG. 141.—Chronic hyperplastic pulpitis (pulp polyp). Lower bicuspid. *C*, carious dentin on the inner wall of the broken-down crown; *E*, stratified squamous epithelium with keratinized surface layer; *P*, hyperplastic pulp tissue; *I*, inflammatory exudate cells in the pulp tissue; *V*, enlarged blood-vessels; *EA*, epithelial attachment to the wall of the root canal. (Boulger, Jour. Dent. Res.)

that permit free proliferation of the hyperplastic tissue. Clinically, the exposed pulp in the latter appears as a red nodule in the pulp chamber. This knob of tissue may vary from the size of a pin-head to that of a pea, and is only slightly sensitive to touch. Upon examination with an instrument it is found that it connects with a strand of living tissue that extends into the root canal. From the similarity of this condition to a polyp of the mucous membrane, this inflammatory hyperplasia of the pulp tissue is called a pulp polyp. Sometimes the surface of the hyperplastic pulp is raw and bleeds easily

during mastication or when touched by an instrument. In other cases the surface is more resistant, being covered with epithelium and having the appearance of gingival tissue. This epithelium originates from the surrounding gingival epithelium, from which it is transplanted in the form of minute cell grafts upon the raw pulp surface. The tissue of a hyperplastic pulp is typical granulation tissue, containing newly formed blood-vessels, fibroblasts, and polyblasts (inflammatory exudate cells).

A case of a hyperplastic pulp with an epithelial covering was reported by Boulger (Fig. 141). In this tooth, a lower bicuspid of an adolescent, the pulp polyp is completely covered by a layer of stratified squamous epithelium, which forms an epithelial attachment to the wall of the root canal. Underneath the hyperplastic part of the pulp, the walls of the root canal have been greatly narrowed by the deposition of calcified masses and secondary dentin. This condition can be considered as an attempt of the pulp to develop a barricade of hard tissue against the advancing infection. The apical portion of the root canal contains normal pulp tissue and, in the upper part, fibrous tissue (granulation tissue) with round cell infiltration and dilatation of the blood-vessels.

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CHAPTER VII.

ACUTE PERIODONTITIS.

PERIODONTITIS or pericementitis is an inflammation of the soft tissue and bone surrounding the root of a tooth. These terms are used in the general description of any inflammatory conditions in this area, regardless of etiology or type of inflammation. In a detailed description of the different clinical types of periodontitis, however, additional terms are used, such as dento-alveolar abscess, granuloma, and cyst.

There are three kinds of periodontitis: traumatic, chemical, and infective. According to the type of inflammation and to the time that has elapsed since the onset of the pathological changes, periodontitis may be divided into acute and chronic periodontitis. This chapter will deal only with acute periodontitis.

TRAUMATIC PERIODONTITIS.

Acute traumatic periodontitis is the reaction of the periodontal tissue to a traumatic injury. The most common causes of it are a blow, kick, or fall upon the face and teeth, injuries during dental operations (forced separation, excessive malleting, injuries during root canal operations), and excessive occlusal stress.

Traumatic Periodontitis Caused by a Blow, Kick, or Fall.—Following a blow, kick, or fall upon the face, one or several teeth are often found loosened and sore to touch or percussion. If the injury is not severe, this soreness usually disappears after a few days without any treatment. An acute injury of this type in human tissues has not yet been studied under the microscope; however, corresponding conditions have been experimentally created in animals and the tissues have been examined microscopically. It was found that the trauma caused multiple tears in the fibers of the periodontal membrane and minute hemorrhages from the periodontal blood-vessels. Immediately following the injury, white blood cells accumulate in the damaged area and aid in the reparative changes by phagocytosis; they remove tissue debris and blood remnants. At the same time, new periodontal membrane fibers are built by the connective tissue cells, and, after a while, the periodontal membrane is completely regenerated and every evidence of the trauma has disappeared.

These reparative processes may take place even if there has been extensive traumatic injury. Teeth which after an accident are so loose that they could almost be extracted with the fingers become firm again and continue to function normally.

Traumatic Periodontitis Caused by Injuries During Dental Operations.—In the practice of dentistry slight traumatic periodontitis is sometimes produced by dental operations. One of the most common causes of a temporary pericementitis is the improper use of the separator. To a certain degree the fibers of the periodontal membrane will yield to the force of tooth separation without injury, but if the separation is continued beyond the limit of tensile strength of the fibers, fiber bundles are torn and crushed, slight hemorrhages occur, and the teeth remain sore for several days. This is especially true if the separating is done while the teeth are anesthetized and the protective action of pain is eliminated. Urban, Beisler, and Skillen reported on forced tooth separation in animals; they found hemorrhages and tearing of the periodontal fiber bundles. These tissue injuries cause a reactive inflammation which, as a rule, terminates in the repair of the damaged tissue. Similar injuries may be caused by excessive malleting during the insertion of foil fillings.

Another type of traumatic periodontitis is caused by injuries during root canal operations. A broach or reamer may pass through the apical foramen and injure the apical periodontal membrane and even the bone beyond the foramen. Clinically, such damage to the periapical tissues is indicated by hemorrhage through the root canal and by soreness of the tooth. A low-grade inflammation develops in the damaged areas; the outcome is either repair and regeneration of the apical periodontal membrane, or, if the injury is complicated by infection, chronic periapical inflammation.

Even the simple removal of a pulp may be followed by a low-grade traumatic periodontitis. This mild form of periapical irritation usually heals, and cementum is later formed over the root end.

Lateral perforation of a root results in traumatic periodontitis. If it occurs in the pulp chamber it can easily be seen; in the root canal it is diagnosed by the unusual amount of hemorrhage from the canal and by radiographic examination. Extensive destruction of the periodontal membrane and bone takes place in the area of injury; leukocytes accumulate around the perforation, and usually a chronic inflammation develops, with formation of granulation tissue and progressive destruction of both soft and hard structures. Such a tooth is usually lost.

Traumatic Pericementitis Caused by Occlusal Trauma.—Traumatic pericementitis may result from biting upon a hard object, such as a stone or a cherry pit. The tooth becomes loose and sore. Microscopic examination of the periodontal tissues reveals the same mild inflammatory changes that are found after a light blow. Usually the symptoms subside in a few days, after repair has taken place in the periodontal membrane. A dental restoration (crown, inlay) that interferes with the normal occlusion may also cause traumatic pericementitis, which persists until normal occlusal relations are re-established. The tissue changes are the same as those described before: tearing and crushing of the periodontal membrane, hemorrhages, and a moderate amount of round cell infiltration.

CHEMICAL PERIODONTITIS.

Chemical periodontitis is caused by the action of drugs that have been introduced into the pulp canal during root canal therapy and that have penetrated beyond the apical foramen. From experimental investigations, it seems that practically every drug used in a root canal causes at least a slight periapical inflammation. The amount of inflammation is in direct proportion to the concentration and penetrating power of the drug, to the amount of drug used, to the length of time the drug is allowed to act upon the tissues, and to the width of the apical foramen. Drugs with great penetrating power will, under otherwise identical circumstances, cause more tissue destruction than drugs with coagulating, and therefore rather self-limiting, properties. In young persons drugs usually act more rapidly and extensively upon the apical tissues than in old individuals with narrow, almost obliterated root canals.

The most common type of toxic periodontitis is that which results from the incorrect application of arsenic trioxide upon the pulp. It has been found experimentally in animals that a slight amount of periapical irritation and inflammation always accompanies the use of arsenic; however, in the first twenty-four or forty-eight hours the changes in the periodontal membrane consist merely of hyperemia and infiltration without actual tissue injury. In this initial stage healing occurs after the drug has been removed. If, however, the arsenic trioxide is permitted to act upon the periapical tissues for a longer period of time, the periodontal membrane is injured beyond repair; the bone becomes involved, and the final outcome is the formation of a chronic periapical inflammation. Acute pericementitis due to arsenic is usually a very painful condition; the

tooth may be extremely sore, and immediate extraction is often the only way to give the patient relief.

The pathology of chemical periodontitis has been the subject of a number of experimental studies. In 1930 Feldmann published a monograph entirely devoted to the subject of chemical periodontitis in animals. He used practically all known drugs and methods of treatment and studied their influence upon the apical tissues over various lengths of time. Feldmann showed that all drugs sealed in the root canal at first caused an inflammatory infiltration of the periapical tissues; after a period of several months the condition of the periodontal tissues varied, depending upon the kinds of drugs used. Roots that had been filled with gutta-percha or oxyphosphate cement following the use of mild drugs were surrounded by normal periodontal tissue, and cementum had been deposited over the apex. In other teeth, in which powerful and penetrating drugs had been sealed, there were progressive inflammation and destruction of the apical hard and soft tissues, without evidence of tissue repair.

Similar experiments were carried out by Coolidge. After removing the pulps from the incisors of dogs, he sealed various drugs in the root canals for a period of twenty-one days. The histological findings revealed considerable variation in the extent and type of reaction. Phenol causes necrosis of the periodontal membrane. The surrounding bone is resorbed. Formo-cresol produces a similar, circumscribed area of coagulation; the periodontal membrane in the vicinity of this necrotic tissue shows the symptoms of a low-grade inflammation with hyperemia of the blood-vessels.

If drugs are used that do not coagulate albumen, such as essential oils or chlorine preparations, the inflammation of the periapical tissues is much more extensive and diffuse. A mild case of diffuse chemical periodontitis is illustrated in Figure 142. Oil of cloves was sealed for twenty-one days in this tooth. At the apical foramen there is a dense inflammatory infiltration that continues uninterruptedly into the adjacent periodontal membrane. A much more severe form of acute periodontitis resulted from sealing eucalyptol, an essential oil, into a dog's tooth for twenty-one days (Fig. 143). The entire periapical region is densely infiltrated with inflammatory round cells; considerable bone resorption and widening of the periodontal space have taken place. Near the center of inflammation at the apical foramen, the periodontal tissue has been completely destroyed and an abscess has developed.

Chlorine preparations produce a reaction of the periapical tissues

similar to that shown in Figure 143. From findings of this kind, however, no conclusions can be drawn as to the advisability of using or discarding certain drugs in dental practice. The main difference between animal experiments and human teeth is that in dogs' teeth intact pulps are removed and the drug placed upon fresh wounds in healthy tissue, while in human teeth pulp tissue and root canal are usually infected; therefore, the use of drugs with bactericidal proper-



FIG. 142.—The beginning of the formation of an acute periapical abscess, produced by sealing eugenol in the root canal of a dog's tooth. *RC*, root canal containing the drug; *A*, abscess formation in the periodontal membrane; *I*, round cell infiltration (leukocytes) in the periphery of the abscess; *V*, hyperemic blood-vessels of the periodontal membrane. (Courtesy of E. D. Coolidge.)



FIG. 143.—Periapical abscess produced by sealing eucalyptol in a dog's tooth. *A*, abscess cavity filled with pus; *R*, extensive resorption of the alveolar bone surrounding the abscess cavity. (Coolidge, Jour. Am. Dent. Assn.)

ties is necessary to control the infection. Although drugs of the latter type may prove harmful to the tissues of dogs, this does not necessarily mean that they should not be used in man, for such drugs may be of value in combating bacteria in infected human root canals and periapical tissues.

INFECTIVE PERIODONTITIS AND DENTO-ALVEOLAR ABSCESS.

Etiology and Pathology of Infective Periodontitis.—Apical periodontitis due to the presence of pathogenic microorganisms is the most common form of periapical inflammation and is the form with which the dentist is most frequently concerned.

Infections of the periapical tissues can be divided into acute and chronic forms. Acute infective periodontitis is characterized by one or several of the cardinal symptoms of inflammation: pain, swelling and redness of the tissues, and fever.

It is impossible to draw a sharp line between periodontitis or pericementitis (inflammation of the periodontium) and alveolar osteitis (inflammation of the alveolar bone). Whenever an infective process has reached the apical foramen and involves the apical periodontal membrane, the microscope reveals changes in the alveolar bone, long before they would be visible in the radiograph. The

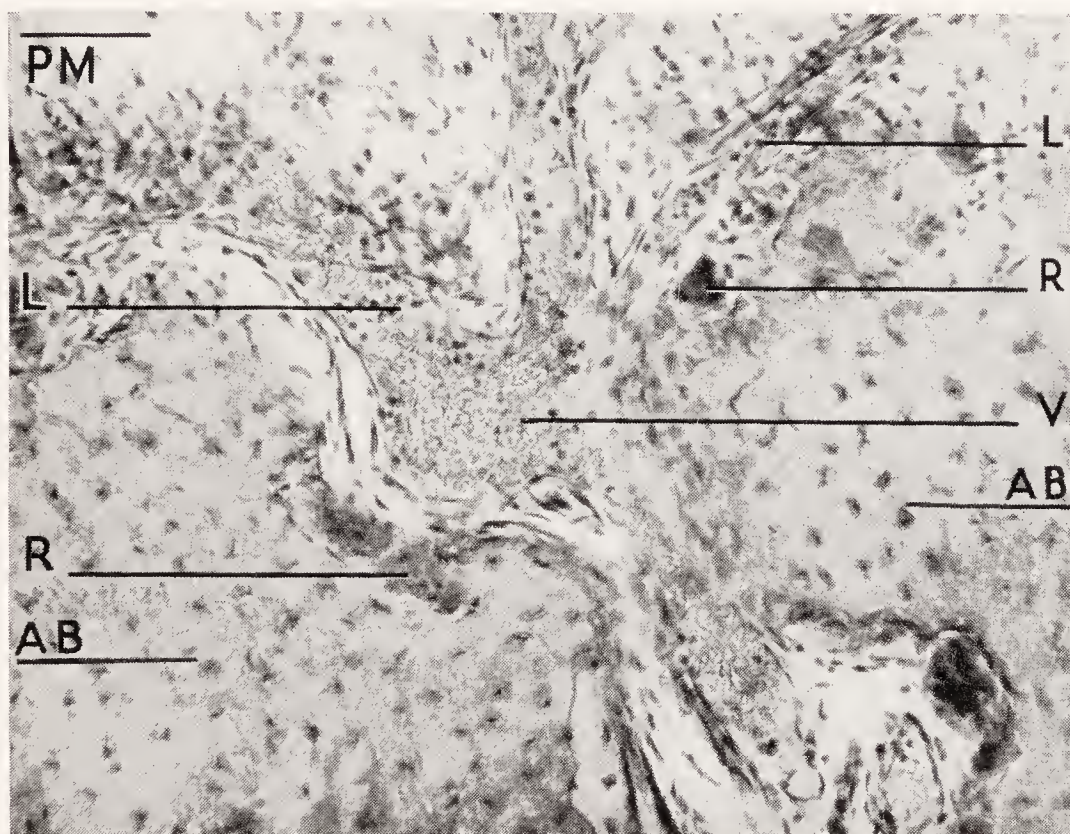


FIG. 144.—The beginning of osteitis of the alveolar bone in acute infective periodontitis. *PM*, periodontal membrane; *V*, hyperemic blood-vessel (vein) leading from the periodontal membrane into the bone; *L*, polymorphonuclear leukocytes in the vessel; *AB*, alveolar bone; *R*, giant cell resorbing the alveolar bone adjacent to the blood-vessel.

blood-vessels in the bone surrounding the inflamed periodontal membrane are hyperemic and contain a large number of polymorphonuclear leukocytes. The alveolar bone in the periphery of the blood-vessels is densely beset with osteoclasts that resorb the bone (Fig. 144). Thus it is impossible to draw a sharp line between inflammation involving the periodontal soft tissues only and that also involving the bone.

Acute infective periodontitis is caused by the presence of pathogenic microorganisms that reach the apical foramen through the pulp canal and spread into the periodontal tissues. In the clinical examination of a tooth with acute periodontitis, it is usually easy to trace the way by which the infection entered the periapical region.

The crown of the tooth is usually destroyed by caries, the pulp is decomposed, and the root canal is empty or filled with débris, or contains an incomplete root canal filling. Infective periodontitis may develop in connection with root canal therapy; then the tooth gradually becomes more and more sensitive and painful following removal of the pulp, until finally typical periodontitis develops with pyogenic microorganisms in root canal and periapical region. Failure to observe a strictly aseptic technique is usually responsible for this kind of periapical infection.

On the other hand, there are times when the etiology of acute periodontitis is not so evident at first glance. There may be the typical symptoms of an acute inflammation of the apical periodontal tissues, looseness and elongation of the tooth, soreness to percussion, and swelling and redness of the soft tissues, and yet the crown may show no caries or other defects. In such a case there is often the history of a trauma (blow, fall), which may have occurred many years before. As a result of this trauma the pulp became necrotic, but no further clinical symptoms developed, except perhaps a slight discoloration of the tooth. However, an acute, purulent inflammation may suddenly develop from such a tooth, seemingly without any exciting cause. Staphylococci and other pathogenic bacteria are found in the pus. It is usually impossible to determine whether these bacteria reached the tooth by way of the blood stream (hematogenous infection) or through minute cracks or defects in fillings or tooth substance (stomatogenous infection).

The first symptoms of infection in the periodontal membrane are the same as elsewhere in the connective tissue, namely, hyperemia of the blood-vessels, slowing of the circulation, migration of white blood cells (polymorphonuclear leukocytes) from within the blood-vessels into the surrounding connective tissue, and diffusion of fluid through the walls of the vessels. Sooner or later, depending upon the virulence of the bacteria and the resistance of the body, the inflamed tissue breaks down, and pus is formed around the root end, a condition known as acute purulent periodontitis. As a rule, the alveolar bone is already involved when the process reaches this stage, and osteoclasts are found in great numbers resorbing the inner lamina of the alveolus and the cancellous bone near it. In this way pus formation and breaking down of tissue spread through the bone, and a purulent alveolar osteitis develops.

Acute inflammation of the apical periodontal membrane causes typical clinical symptoms. The first reactions of the periodontal soft tissues to the invasion of bacteria are hyperemia, edema, and

leukocytic infiltration. These changes cause an increase in the volume of the tissues, and since the periodontal membrane is enclosed between bone and cementum and cannot expand, pressure is created within the apical periodontal space. The tooth appears slightly elongated, for it protrudes from the alveolus as a result of the increased tissue tension at the root end; also it is loosened because the fibers of the periodontal membrane are edematous. It is sore, either spontaneously or on percussion and during mastication, since the nerves in the periodontal membrane are irritated by the inflammatory process. However, not all symptoms of acute apical infection, elongation, loosening and soreness, are necessarily present in every case. If a tooth is extracted in this stage of acute periodontitis, the hyperemia of the periodontal membrane is indicated by the redness of the soft tissue that adheres to the apex of the extracted root.

After the infiltration of the periodontal membrane has reached a certain degree, the soft tissues begin to break down. Pus accumulates in the periodontal space and in the marrow cavities of the bone surrounding the apical foramen. In this stage the tooth is often very sore, and continuous pulsating pain indicates the presence of a purulent process in the depth of the alveolus.

An important symptom is the change in the regional lymph nodes, which are located at the lower border of the mandible. The anterior group, the submental lymph nodes, are connected with the lymphatics draining from the lower anterior teeth; the posterior group, the submaxillary lymph nodes, are connected with the upper and lower bicuspid and molars. During acute purulent periodontitis these lymph nodes are usually swollen and tender.

If in this stage of the inflammatory process drainage is established through the root canal by opening into the tooth and removing the débris and root filling material, pus is sometimes discharged through the root canal, after which the patient has immediate relief from the throbbing pain. The same relief is obtained if the tooth is extracted; then the purulent exudate can drain freely through the alveolus and the inflammation subsides in a short time.

If drainage is not established, the purulent inflammation burrows through the bone and soon reaches the outer surface of the alveolus. Here the spread of this inflammatory process is usually temporarily halted by the resistant layer of periosteum that covers the outer surface of the bone. This stage is spoken of as subperiosteal abscess.

Acute Dento-alveolar Abscess.—As the purulent inflammation approaches the surface of the bone, a new and important symptom

is observed, namely, edema and swelling of the soft tissues of the face. This condition is known as cellulitis. It usually reaches its height when the pus has perforated the outer lamina of the alveolar process and appears under the periosteum.

If the inflammatory process starts from an upper anterior tooth, the upper lip frequently becomes greatly swollen, stiff, and distended. An acute dento-alveolar abscess originating from an upper bicuspid or molar causes cellulitis of the cheek; frequently the lower eyelid is edematous so that the patient is unable to open his eye on the affected side. If a lower anterior tooth is involved, the lower lip and the chin region are swollen; from a lower posterior tooth, the swelling extends over the lower part of the cheek, the angle of the mandible, and sometimes downward into the submaxillary region.

After the pus has perforated the outer plate of the alveolar process, it spreads along the bone surface, separating the periosteum and the mucous membrane from the bone. This mucoperiosteum is elevated by the pressure of the underlying pus and fluctuates when palpated with the finger. The lymph nodes at the lower border of the mandible are swollen and tender. There is a rise in temperature; headache, constipation, and general malaise often accompany such acute purulent processes in the jaw.

Finally, the pus forces its way through the overlying soft tissues and appears on the surface; or the swelling is incised, the abscess is opened, and pus is discharged. With the free drainage of the pus, the acute dento-alveolar abscess usually subsides within a short time; pain, swelling, and fever disappear, and the patient is well again. The final outcome is either transition from the acute into a chronic inflammatory process or healing after extraction of the tooth. By far the largest number of acute dento-alveolar abscesses develops as an acute flare-up of a chronic periapical inflammation of long standing; after the acute symptoms have subsided, chronic periodontitis persists, forming a potential source of new acute attacks.

TOPOGRAPHY OF ROOT ENDS AND SURROUNDING STRUCTURES.

The place where the pus perforates in an acute dento-alveolar abscess depends upon the topographical relation between root end and surrounding structures. As a rule, the pus makes its way through the jaw in the direction of least resistance; it appears on that side of the jaw where the bone between root end and outer soft tissues is thinnest. It is therefore important to know the anatomical

variations in the relationship between the apices and the adjacent tissues and structures.

Maxillary Teeth.—The apices of the upper incisors, cuspids, and the buccal roots of upper bicuspid and molars are located close to the thin buccal bone plate of the alveolar process. Pus originating from apical infection in these teeth is therefore most likely to burrow through the thin labial or buccal plate of the alveolar process and appear in the vestibule. Sometimes, in individuals with low alveolar processes and long roots, an inflammatory process at the apex of an upper central incisor perforates into the floor of the nose, causing an abscess inside the nasal opening.

The apex of the upper lateral incisor is sometimes located toward the lingual side of the other anterior teeth, lying close to the lingual surface of the alveolar process. An acute dento-alveolar abscess originating in this tooth may, therefore, erupt toward the palate and form a palatine abscess. Because of the rigidity and resistance of the palatine soft tissues, an acute dento-alveolar abscess in the palate frequently persists for a considerable length of time before perforation takes place; the pressure of the subperiosteal abscess may detach much of the palatine periosteum from the bone. This is especially true of abscesses originating from the lingual roots of upper molars, which often have a tendency to spread under the periosteum back toward the soft palate instead of perforating into the oral cavity. In a palatal abscess there is usually no cellulitis of the face.

The upper cuspid has a long root, the end of which is sometimes located in a higher level than the vestibule. An inflammatory process originating from the apex of this tooth may extend upward into the canine fossa and then perforate through the skin below the inner angle of the eye. In chronic periodontitis of the cuspid, a sinus may develop in this area, which may be confused with a fistula of the naso-lacrimal duct.

Relationship Between Maxillary Teeth and Maxillary Sinus.—In acute dento-alveolar abscesses originating from upper bicuspid and molars, the relationship between these teeth and the maxillary sinus (antrum of Highmore) must be considered. The maxillary sinus is a large cavity, shaped approximately like a four-sided pyramid. The apex of this pyramid points laterally (outward) and is located within the zygomatic process of the maxilla. The base points medially (inward) and is formed by the lateral wall of the nose. The roof or superior (orbital) wall is formed by the lower wall of the orbit; it contains the infraorbital vessels and nerves. The poste-

rior or pterygo-maxillary wall is concave; it is formed by the posterior bone plate of the maxilla. The antero-lateral or facial wall lies below the eye and behind the cheek; it is formed by the anterior and lateral bone plates of the maxilla. The nasal wall separates the antrum from the middle and inferior meatus of the nose; in it is located the normal opening or ostium through which the sinus communicates with the middle meatus of the nose. The floor of the

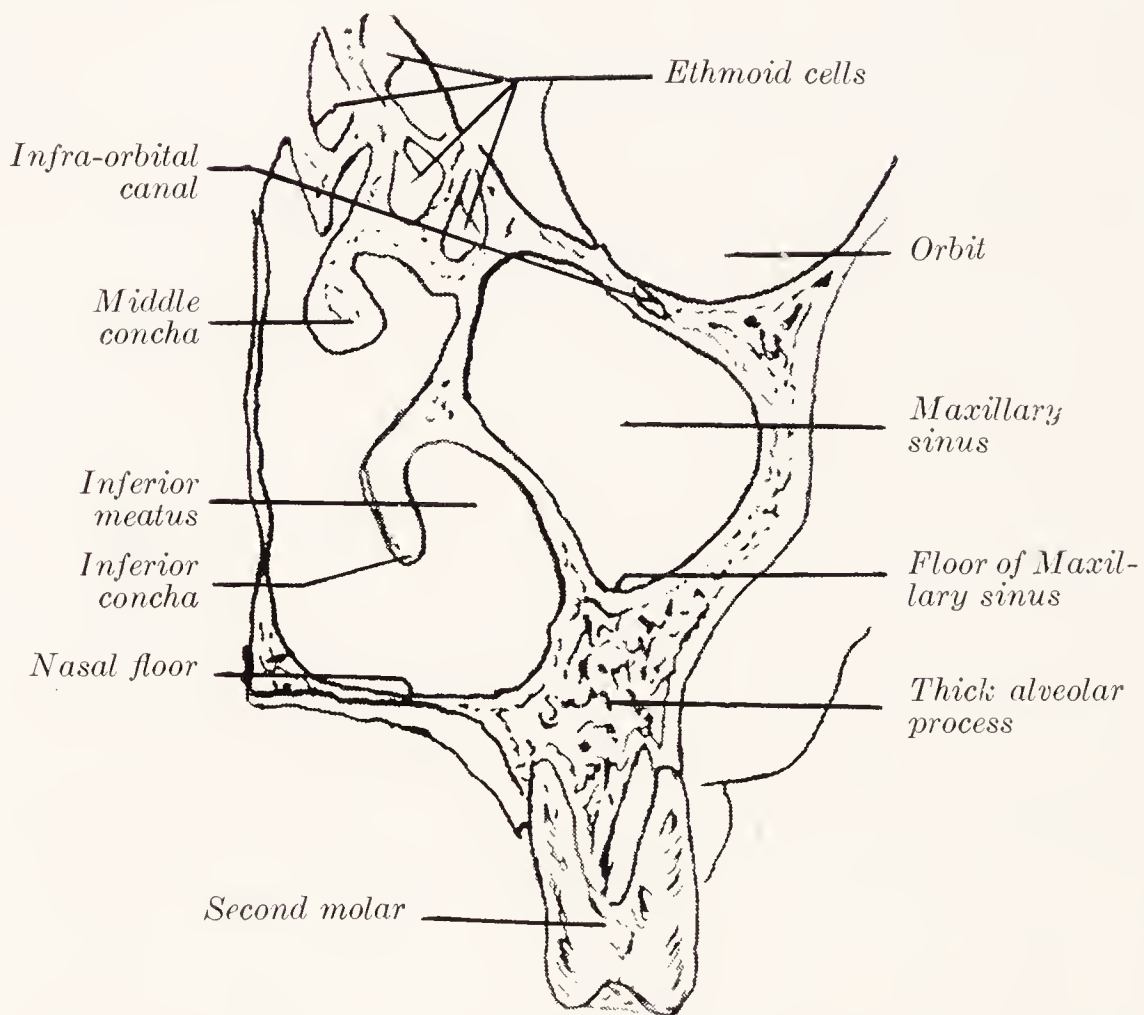


FIG. 145.—Diagram showing high maxillary sinus floor and thick alveolar process. The floor of the antrum is above the floor of the nose. (Mustian, Jour. Am. Dent. Assn.)

antrum or dental wall is formed by the hard palate and the alveolar bone surrounding the bicuspid and molars; it is uneven and sometimes subdivided by bony septa.

The cavity of the sinus is lined with the typical ciliated columnar epithelium of the upper respiratory tract. Most diseases of the sinus consist of an infection and inflammation of this mucous membrane. In approximately 80 per cent of all antral disease, the infection gains access from the nose through the natural ostium; the remaining 20 per cent are of dental origin, reaching the antrum through its floor.

The size of the antrum varies greatly. It is small in youth and gradually becomes larger, until in old age the bony walls may be

reduced almost to paper thinness. In adults the capacity varies from 9.5 to 20 cc. Often the size of the sinus cavities in one skull varies considerably.

A large antrum may extend anteriorly to the alveolus of the cuspid and posteriorly well into the maxillary tuberosity. In the vertical dimension these variations are of still greater dental significance. If the antrum is small, the root ends may be separated from the antral floor by a thick layer of bone. If the antrum is large, the root ends of one or several teeth, usually the first and second molars, extend into the sinus floor as conical projections, sometimes covered

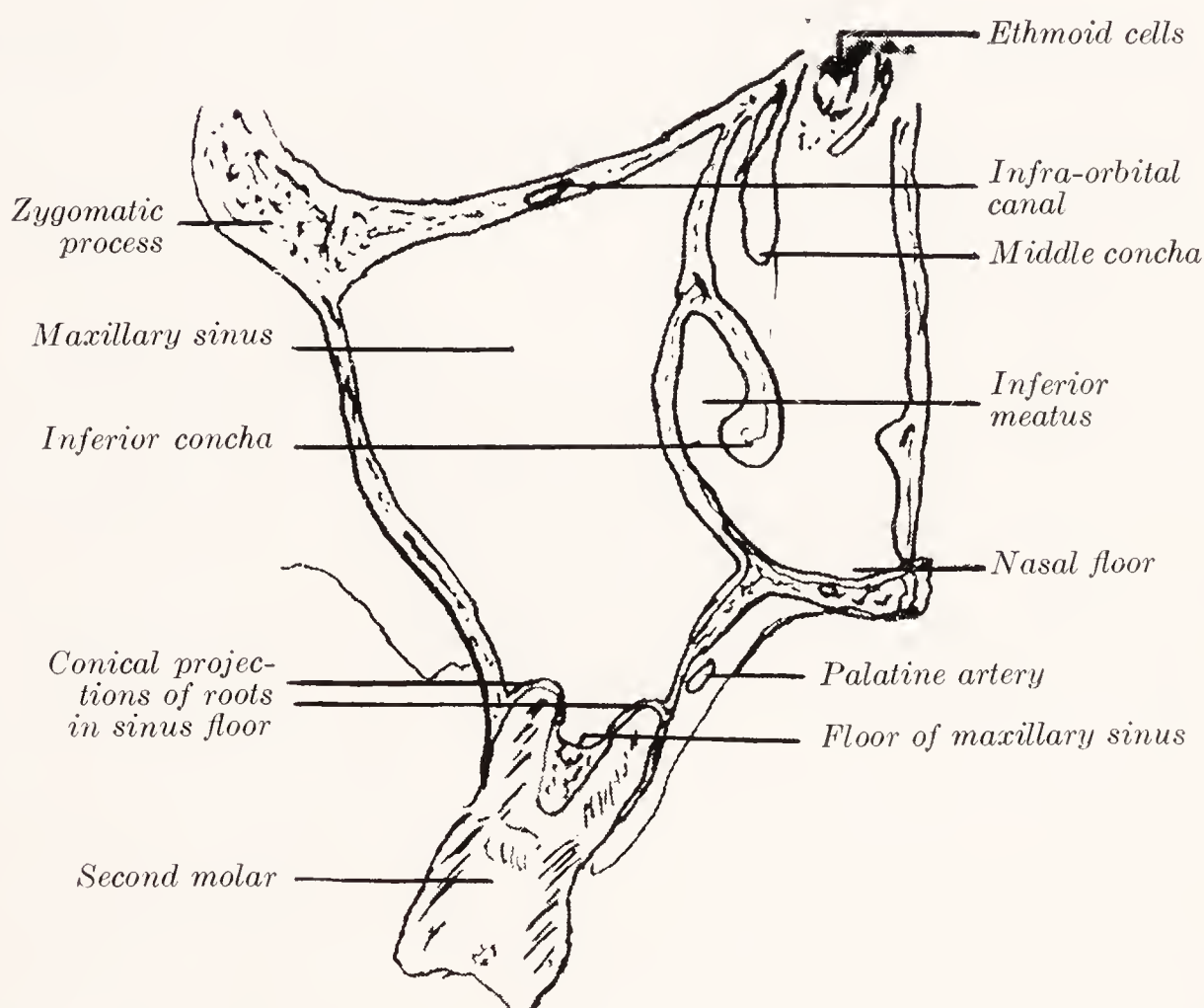


FIG. 146.—Diagram showing large maxillary sinus with a low floor extending between the roots of the second molar. The floor of the antrum is below that of the nose. (Mustian, Jour. Am. Dent. Assn.)

only by the mucoperiosteum (Figs. 145 and 146). A large maxillary sinus may have pocket-like extensions downward between the upper teeth or into the bifurcation of the upper molars. Under such conditions, if one of these teeth is extracted, there may be a communication between oral cavity and maxillary sinus through the empty socket. By careless attempts to remove teeth or roots, a tooth or apex may be pushed upward into the sinus.

The participation of the maxillary sinus in dental inflammation depends largely upon the size of the antrum and its relationship to

the teeth. If the roots are close to or rise above the floor of the sinus, the latter is likely to be involved. The thin layer of bone and muco-periosteum on the floor of the antrum is destroyed, and pus and granulation tissue from the diseased tooth spread into the antrum (Fig. 147). If, on the other hand, the floor of the sinus is far above the root ends and the latter are close to the buccal bone surface, a dento-alveolar abscess is most likely to erupt into the vestibule.

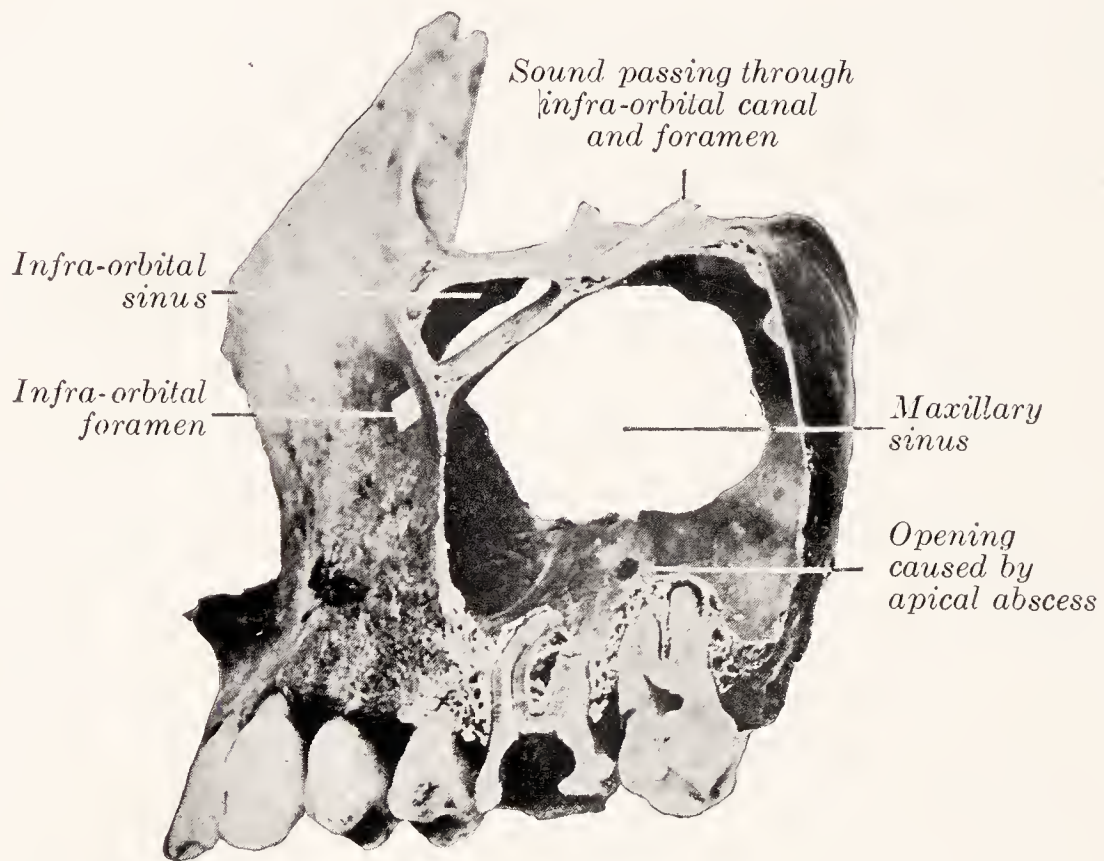


FIG. 147.—Antero-posterior cut through the maxilla, showing the opening of a dental abscess into the maxillary sinus. (Cryer.)

Any involvement of the maxillary sinus of dental origin requires coöperation with a competent physician; the dentist alone is not qualified to deal with a complication of this kind.

Mandibular Teeth.—In the lower jaw the relation of the root ends to the surrounding structures depends upon the length of the roots, the height of the alveolar process, and the location of the attachment of the tissues to the outer and inner surface of the mandible. Acute dento-alveolar abscesses originating from lower teeth usually erupt into the labial or buccal vestibule. The soft tissues overlying the mandible are inflamed and swollen, and there is a cellulitis of lower lip, chin, or cheek. Occasionally the pus perforates toward the lingual side and appears in the floor of the mouth.

A not uncommon complication of an acute dento-alveolar abscess in a lower tooth is the perforation of the pus through the skin along

the lower border of the mandible. This is particularly observed in acute dento-alveolar abscesses developing from the lower incisors or first molars of young individuals. These teeth frequently have long roots that are deeply implanted in the mandible; the apices are closer to the lower border of the mandible than to the buccal or lingual surface. Consequently, pus developing at the root ends is likely to burrow through the bone at the lower border. Then the pus perforates the periosteum and the muscular layer and finally breaks through the skin spontaneously, or it may be drained through an incision in the skin. If the infected tooth is not extracted, a chronic sinus draining through the skin is likely to develop. After extraction, frequently a depressed, contracted scar remains, which adheres to the underlying bone and later may require correction by a plastic operation.

OSTEOMYELITIS DEVELOPING FROM ACUTE DENTO-ALVEOLAR ABSCESS.

A serious complication of an acute dento-alveolar abscess is osteomyelitis of the jaw bone. Osteomyelitis is an acute or chronic infection of the bone and bone-marrow by pus-producing microorganisms. In the course of an acute osteomyelitis the periosteum and endosteum of the bone are detached from the bone surface and destroyed, and as a result the bone loses its blood supply and dies. After the acute inflammatory process has come to a standstill, granulation tissue and osteoclasts separate the dead from the living bone. The detached piece of dead bone is called a sequestrum; it is finally discarded, and then the osteomyelitis terminates.

The clinical signs of an acute osteomyelitis as a result of an acute dento-alveolar abscess are increasing severity of the symptoms, extensive cellulitis, pain, high fever, rapid pulse, and leukocytosis. Instead of perforating the soft tissues, the pus spreads along the bone surface and inside the marrow cavities, detaches the periosteum, and destroys the vitality of the bone. Gradually the acute symptoms subside, and a long period of suppuration ensues, until the dead, denuded bone has been demarcated and can be eliminated. The size of the lost portion varies from the alveolar process of one or two teeth to most of the mandible or maxilla.

Three fundamental factors are involved in the development of osteomyelitis, namely, high virulence of the infection, low resistance of the patient, and lack of drainage. The last point is important since it is the one most amenable to therapy. Whenever in the

course of an acute dental infection the abscess fails to point, but instead shows a tendency to spread along or through the bone, incisions should be made to establish drainage and thus to localize and terminate the infection.

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CHAPTER VIII.

CHRONIC PERIODONTITIS.

CHRONIC inflammation of the apical periodontal tissue is characterized by the presence of a chronic osteitis with transformation of periodontal membrane and alveolar bone into granulation tissue. To facilitate the understanding of the morbid anatomy of chronic periodontitis, the most important facts about granulation tissue will be reviewed briefly.

STRUCTURE AND SIGNIFICANCE OF GRANULATION TISSUE.

Granulation tissue is the product of a chronic inflammation. Its formation is a defensive or reparative reaction of the organism, inasmuch as in many instances granulation tissue initiates the healing of an infection or wound.

A typical instance of the development of granulation tissue is the healing of a large, open wound on the body surface. A few days after the epithelium has been lost, the raw surface is covered with a red tissue with uneven, finely granulated surface, from which it derives its name. Soon the entire wound surface is covered by this new tissue, and the epithelium from the margins of the wound begins to grow over it. Subsequently granulation tissue becomes transformed into scar tissue.

Granulation tissue is young, very vascular connective tissue consisting of fibroblasts, numerous capillaries, inflammatory exudate cells, and occasionally other cell forms, such as giant cells.

Fibroblasts.—Fibroblasts are connective tissue cells with pale, oval nuclei containing fine, dust-like chromatin and several nucleoli. The protoplasm of the fibroblasts has spear-shaped processes that extend from one cell to another. The fibroblasts produce a fibrous intercellular substance, the connective tissue fibrillæ, which, together with the cells, form a fine network that is the framework of every soft tissue of connective tissue type.

The fibroblasts in granulation tissue develop by mitosis from the preëxisting fibroblasts in the inflamed or injured area; in a skin wound, for instance, new fibroblasts are derived from the fibroblasts in the subcutaneous connective tissue in the vicinity of the wound. In a chronic inflammation of the alveolar bone, the fibroblasts in

the granulation tissue develop from the periodontal membrane and from the connective tissue of the periosteum and endosteum.

The fibroblasts do not, in a strict sense, take part in the inflammatory process. Their function is one of repair and regeneration; they incapsulate the inflamed area, replace lost tissue, and form scar tissue.

Capillaries.—Granulation tissue contains a great number of capillaries, which give it its typical red color. These capillaries develop from the blood-vessels in the surrounding tissue. From these blood-vessels solid sprouts of proliferating endothelial cells invade the forming granulation tissue; later they are transformed into true capillaries by being hollowed out from within (Fig. 148).

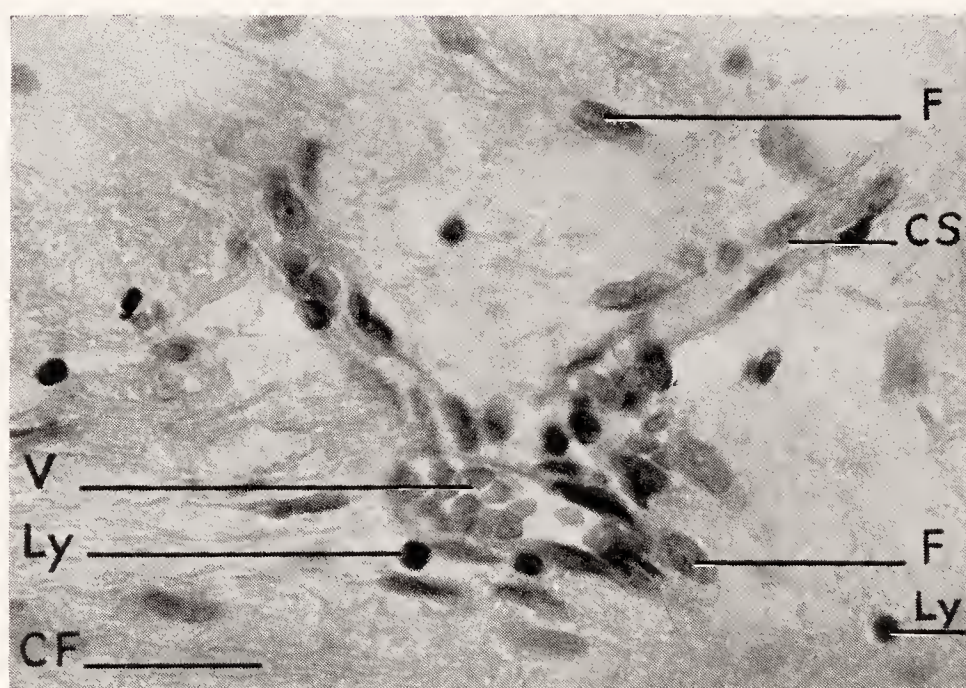


FIG. 148.—Capillaries in granulation tissue in the oral cavity. *V*, capillary blood-vessel containing erythrocytes; *CS*, capillary sprout growing from the vessel into the surrounding granulation tissue; *F*, nuclei of fibroblast; *CF*, connective tissue fibrillæ; *Ly*, polyblast (lymphocyte).

Exudate Cells.—The capillaries are the pathways by which the body's main cells of defense, the white blood cells, reach the area of injury or infection. Here these cells overcome the infection and dispose of any tissue débris that may be present. The inflammatory exudate cells are an important element in the granulation tissue. The usual terms "round cells" or "round-cell infiltration" which are applied to these cells merely indicate that they have a more rounded form than the fixed connective tissue cells. It is necessary to study them under high magnification and to consider their origin in order to understand their significance. Two main types of inflammatory round cells can be differentiated, namely, leukocytes and polyblasts.

Leukocytes.—Normally, neutrophil granular leukocytes with lobulated nuclei (polymorphonuclear leukocytes) are always found in

the blood stream and there constitute from 65 to 75 per cent of the total amount of white blood cells. Their nuclei consist of several nuclear divisions held together by delicate chromatin bridges. They develop in the bone-marrow from the myelocytes. The polymorphonuclear leukocytes are not found in normal connective tissue. However, in areas of injury or infection they accumulate in the small blood-vessels; they leave the center of the blood stream, adhere to the vessel wall, and finally migrate through the walls of the capillaries and into the surrounding connective tissue (Fig. 149). Polymorphonuclear leukocytes are especially numerous in the early stages of inflammation and in acute suppuration; they are the main

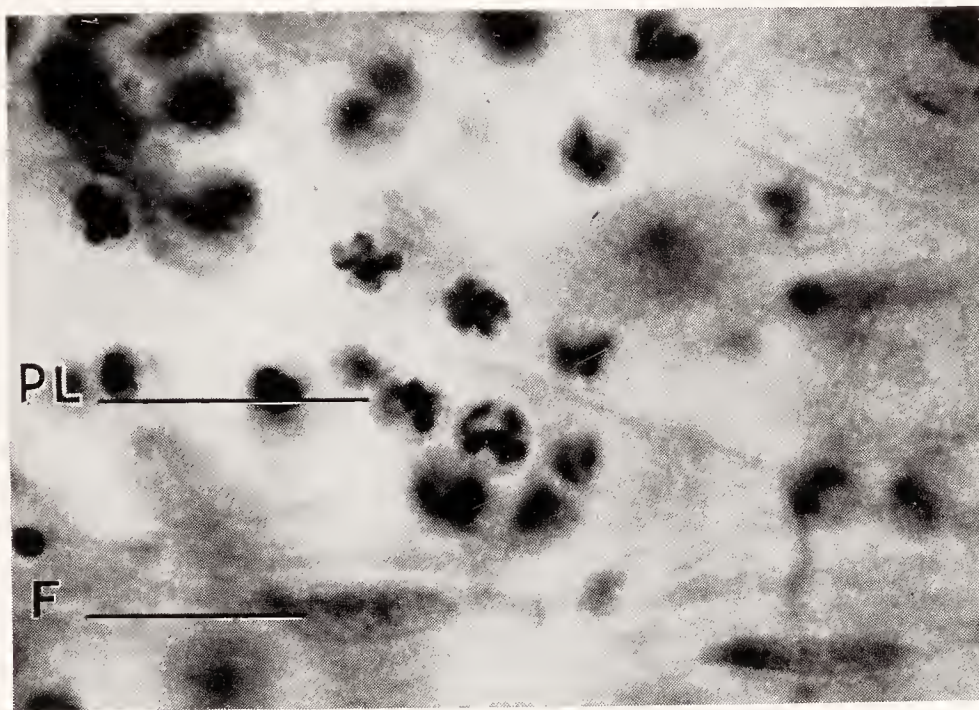


FIG. 149.—Typical cell forms in acute purulent inflammation of the periapical tissue. Infiltration of the connective tissue with polymorphonuclear leukocytes, *PL*, with round protoplasmic bodies and lobular nuclei; *F*, fibroblast.

cellular constituents of pus. In the later stages of inflammation, the leukocytes degenerate and disappear. For this reason they are not very numerous in granulation tissue, since the latter is the result of chronic inflammation of long standing.

The polymorphonuclear leukocytes form the first line of defense of the body against infection. Their function is the destruction of the pathogenic microorganisms in the inflamed tissue. This is performed by phagocytosis and intracellular destruction of the bacteria. The leukocytes engulf the living bacteria, and by means of special ferments dissolve and destroy the bacterial bodies. Many leukocytes die in the inflamed area, and by their disintegration liberate a proteolytic ferment that dissolves tissue débris and thus aids in its ultimate removal.

Polyblasts.—Maximow, who made a very thorough study of the development and morphology of the various cell types involved in

inflammatory reactions, called all inflammatory exudate cells, with the exception of the polymorphonuclear leukocytes, "polyblasts," thus indicating their different shape and origin. These cells have a double origin: they are derived either from the connective tissue or from the non-granular leukocytes (lymphocytes and monocytes) of the blood. The histogenous polyblasts develop from the histiocytes or resting wandering cells of the loose connective tissue. As a result of inflammation, the histiocytes are transformed into large cells with ameboid motility; they migrate toward the center of inflammation where they become phagocytes and engulf bacteria and cell débris. The hematogenous polyblasts originate from the lymphocytes and monocytes of the blood. In case of inflammation these blood cells, together with the polymorphonuclear leukocytes, migrate through the walls of the vessels and appear in the connective tissue; but whereas the polymorphonuclear leukocytes disappear in the later stages of inflammation, the lymphocytes persist and in chronic inflammation are the main cells of the exudate. They are often described as "small round cells." The nucleus of the lymphocyte is spherical; the scanty cytoplasm is confined to a thin layer around the nucleus.

According to Maximow, the endothelial cells of the capillary walls do not contribute to the formation of exudate cells. The endothelial cells of the adult organism are highly differentiated cell elements that cannot be transformed into polyblasts. The latter are derived wholly from the two sources already mentioned, the histiocytes and the white blood cells.

Polyblasts are always found in large numbers in granulation tissue. They appear in different forms and sizes. Some have simple, round nuclei similar to those of lymphocytes; others have oval or kidney-shaped nuclei. The so-called plasma cells are also a form of polyblasts. Plasma cells are characteristic of some types of chronic infections and granulation tissue; they are easily recognized by their large oval or triangular body of cytoplasm and round nucleus located in an eccentric position in the periphery of the cytoplasm. Because the chromatin granules are arranged in the periphery, the nucleus of a plasma cell has a design similar to a wheel with spokes ("cart-wheel nucleus," Fig. 150). In chronic inflammations of the oral and dental tissues plasma cells are frequently present in large numbers.

Polyblasts, which are phagocytic cells, have the property of ameboid movement and migrate actively through the tissues. If they encounter bacteria or remnants of tissue decomposition, such as dead

cells and fat granules, they engulf and digest them in their bodies. A typical example of phagocytosis is shown in Figure 151, which was taken of an area of granulation tissue in the oral cavity. A minute hemorrhage had occurred in this tissue some time previous to the removal of the specimen. Two large oblong cells are found in the

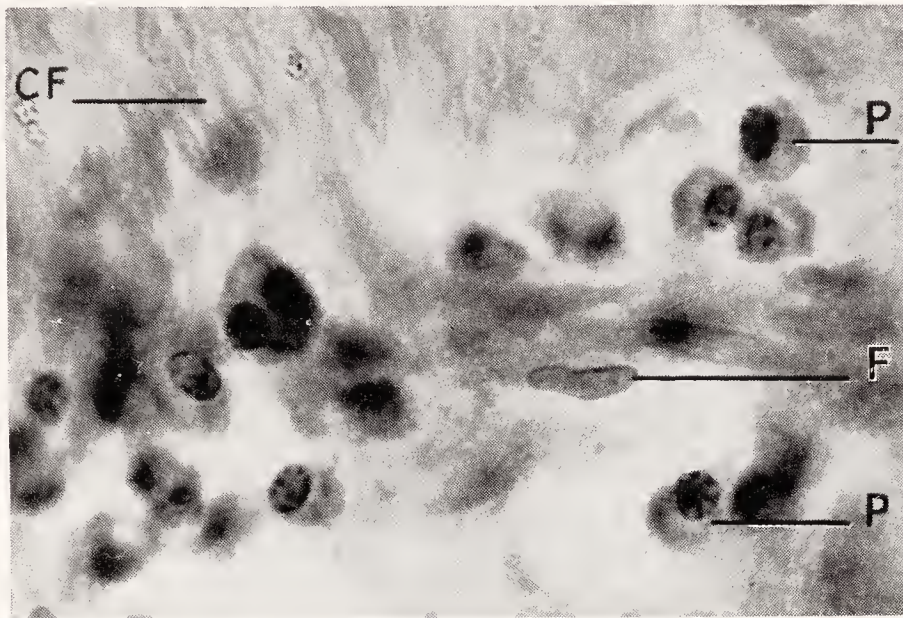


FIG. 150.—Typical cell forms in chronic inflammation of the periapical tissue. *P*, plasma cells (polyblasts) with “cartwheel” nuclei; *F*, fibroblast; *CF*, connective tissue fibrillæ.

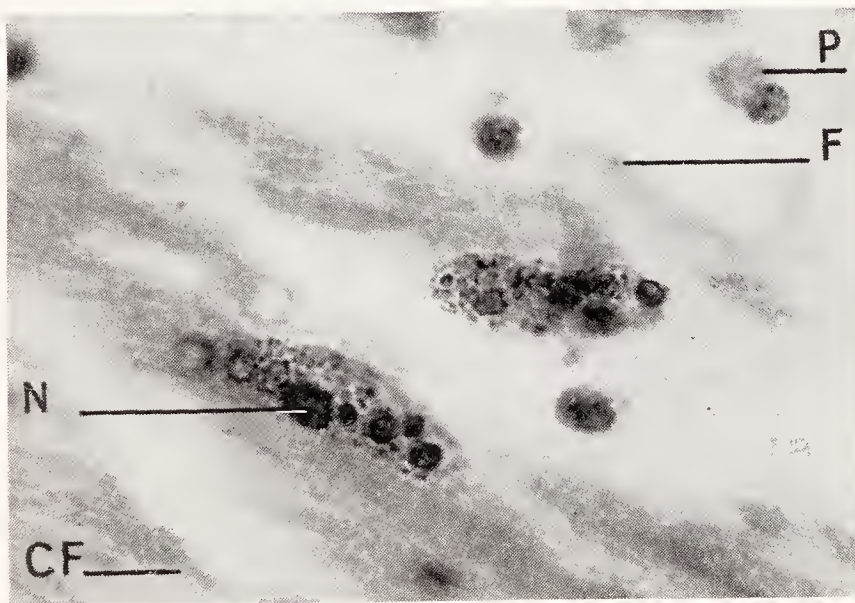


FIG. 151.—Phagocytosis in granulation tissue in the oral cavity. Two large phagocytic polyblasts, the cytoplasm of which contains a large number of granules of blood pigment from a nearby small hemorrhage. *N*, nucleus of the polyblast; *F*, fibroblast; *P*, polyblast (plasma cell); *CF*, connective tissue fibrillæ.

connective tissue near the center of hemorrhage; their cytoplasm contains bright yellowish-brown granules of hemosiderin (blood pigment) which were taken up by the phagocytes. The nuclei of these cells are faint among the yellowish-brown masses. From the form of the cells and their nuclei, it is probable that they originally were polyblasts of the plasma cell type.

If an abscess cavity is present in the center of the granulation tissue, polyblasts from the surrounding granulation tissue are discharged into it. The pus phagocytes in a chronic abscess are derived partly from the monocytes and lymphocytes of the capillaries and partly from the histiocytes in the surrounding fibrous tissue capsule.

After the inflammation has subsided, some of the polyblasts may be transformed back into resting wandering cells (histiocytes) and finally into fibroblasts, thus taking part in the formation of scar tissue.

Giant Cells.—Giant cells are sometimes found in granulation tissue. In an epulis or in tuberculous granulation tissue, for instance, giant cells are abundant; in granulation tissue around infected root ends, they sometimes line the surface of the surrounding bone as osteoclasts but are not found within the granulation tissue itself. Giant cells are probably derived from connective tissue and osteogenic cells by continued reproduction of the nuclei without subsequent division of the cytoplasm, or they develop by a fusion of polyblasts; thus very large cells result, with one protoplasmic body and many nuclei. The function of giant cells is the elimination of material that is difficult to remove, such as elastic fibers, blood pigment, foreign bodies, and calcified tissues.

CHANGES IN GRANULATION TISSUE IN INCREASED OR DECREASED INTENSITY OF INFLAMMATION.

Elements of Granulation Tissue: Fibroblasts, Connective Tissue Fibrillæ, Capillaries, Polyblasts (Lymphocytes, Plasma Cells, Various Phagocytes); Occasionally Leukocytes, Giant Cells.

<i>A.</i>	<i>B.</i>
Acute exacerbation of inflammation:	Decrease of inflammation:
Increase in number of polymorphonuclear leukocytes.	Reduction in number of polyblasts.
Destruction of connective tissue elements.	Reduction in number of capillaries.
Formation of pus, consisting of leukocytes, cell débris, and exudate.	Increase in number of fibroblasts; formation of connective tissue fibrillæ.
Abscess formation.	Scar formation.

This table gives a schematic outline of the changes that granulation tissue may undergo under various conditions. Two practical examples will be given to illustrate conditions *A* and *B* of the table.

A, acute exacerbation of a chronic inflammation: An acute dento-alveolar abscess with all the clinical symptoms of an acute purulent inflammation develops from a chronic inflammatory condition (granuloma). The connective tissue elements in the granulation tissue break down, and an abscess cavity filled with pus (leukocytes, bacteria, and cell débris) is formed.

B, decrease of inflammation: A tooth with an infected root canal and chronic periapical inflammation (granuloma) is extracted; the granulation tissue remains in the socket. Within the next few days after the removal of the source of infection, the fibrous elements in the granulation tissue proliferate, the polyblasts disappear, and a connective tissue scar develops. Subsequently bone is formed in the fibrous tissue of the scar.

The conditions described under *A* and *B* are sometimes found simultaneously in one granuloma. In the periphery farthest from the tooth, scar tissue may be present, while toward the center there is an increase in inflammatory cells (polyblasts); at the apex the tissue has broken down and pus is forming. Thus, the tissue is prepared for all possible events. If the inflammation increases, the small area of pus around the apex spreads, and a large abscess cavity finally takes the place of the former granulation tissue; if, on the other hand, the infection in the root canal is controlled, the proliferation of connective tissue elements advances from the periphery toward the center until all of the granulation tissue is transformed into scar tissue and ultimately into bone.

HISTOPATHOLOGY OF THE MANIFESTATIONS OF CHRONIC PERIAPICAL INFLAMMATION.

Granuloma.—The simplest form of chronic periapical inflammation consists of a transformation of the apical periodontal membrane and alveolar bone into granulation tissue. This condition is known as dental granuloma. A granuloma may vary in size from a pin-head to 8 or 10 mm. or even larger, depending upon the length of its duration and the intensity of the inflammation.

The earliest changes in chronic periapical inflammation usually take place while the pulp canal still contains some vital pulp tissue in a state of chronic pulpitis. An example may illustrate this. In the microscopic examination of a human lower molar that had been extracted because of pulpitis, the pulp reveals extensive inflammatory changes (Fig. 152). In the mesial pulp horn, a large abscess has developed beneath the floor of a deep cavity; this abscess is distinctly separated from the surrounding pulp tissue, which is characteristic of a chronic inflammation of long standing. In the distal portion of the pulp chamber, the pulp tissue shows diffuse infiltration and enlargement of the blood-vessels; on the distal wall, odontoblasts are still present. The microscopic diagnosis of this condition would be chronic pulpitis with pulp abscess.

The question is whether or not the periapical tissues are already involved. Clinically such teeth are frequently sensitive to percussion, which indicates an inflammatory edema of the periodontal



FIG. 152.—Chronic pulpitis with formation of a pulp abscess under a deep cavity in a lower second molar. *FC*, floor of cavity; *A*, pulp abscess; *I*, diffuse inflammatory infiltration of the entire pulp; *Od*, odontoblasts on the distal wall of the pulp chamber; *V*, enlarged blood-vessels in the distal root canal.



FIG. 153.—Mesial root end of the tooth shown in Figure 152. Dense accumulation of inflammatory exudate cells (polyblasts) at the apical foramen. *AF*, apical foramen; *I*, inflammatory round cells; *PM*, fibrous connective tissue of the periodontal membrane.

membrane. The microscopic examination of the root ends reveals a circumscribed area of round-cell infiltration in the periodontal membrane next to the apical foramen (Fig. 153). The inflammatory cells are polyblasts, mostly plasma cells, indicating an inflammation

of long standing. This inflammatory area at the root end is still too small to be visible radiographically; it is merely the first step in the gradual development of a more extensive periapical inflammation. If the inflammation and destruction of the pulp had advanced rootward, the periapical inflammation would have increased; gradually it would have extended beyond the periodontal membrane and involved the alveolar bone. This specimen is additional evi-

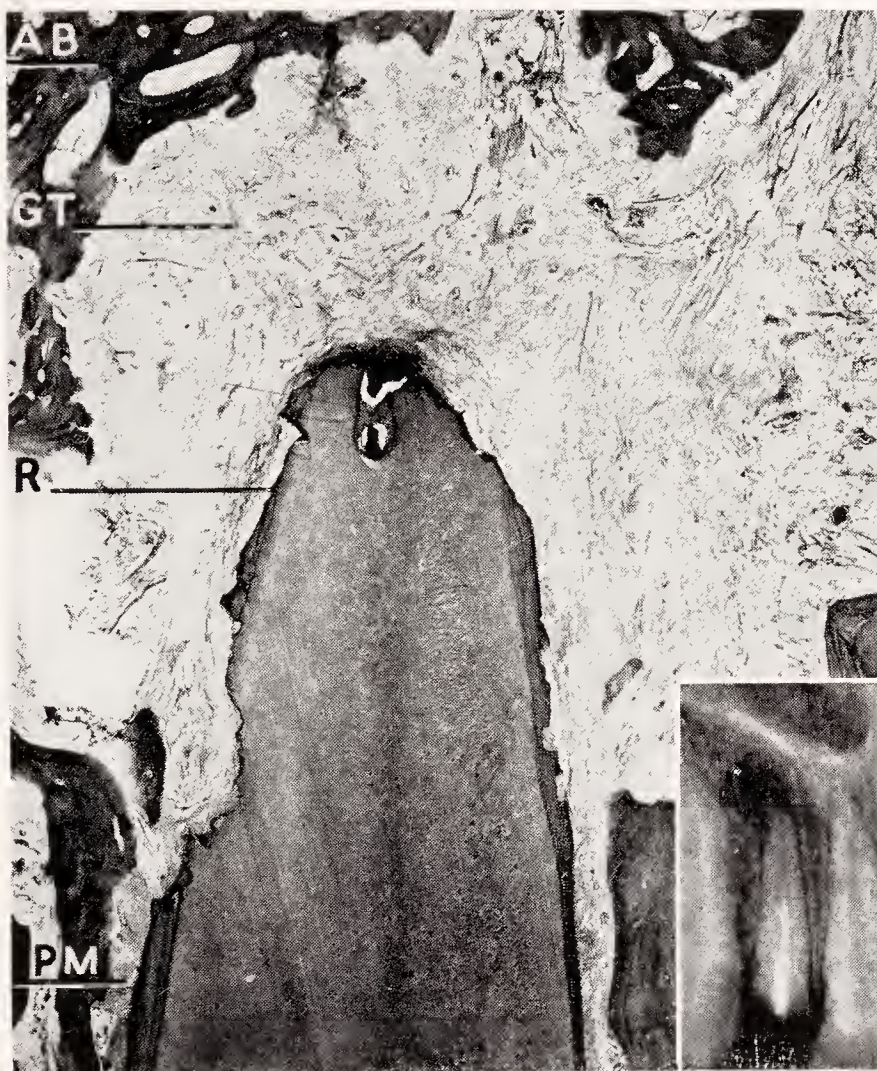


FIG. 154.—Solid granuloma. Mesio-distal section through the root end of an upper lateral incisor with decomposed pulp and imperfect root canal filling. The radiograph of the specimen taken before decalcification is inserted in the lower right corner. Because of a slight apical curvature of the root, the root canal is visible only at the apical foramen. *AB*, alveolar bone; *GT*, granulation tissue occupying the space between alveolar bone and root; *R*, resorption of the root surface; *PM*, normal periodontal membrane.

dence that neither clinically nor microscopically is there a sharp dividing line between pulpitis and periodontitis.

A few typical cases of apical granuloma will be described here. These specimens were obtained by autopsy from human jaws. Since the radiograph is commonly used in dental practice to diagnose chronic periapical inflammation, a radiograph of the specimen accompanies each microscopic picture.

Simple Granuloma.—Figure 154 illustrates a mesio-distal section through the root end of an upper lateral incisor. The radiograph

shows an incomplete root canal filling. The apex is surrounded by a sharply outlined area of bone destruction, and the root end projects into this radiolucent area. The rest of the periodontal space appears intact. The area around the root end is filled with granulation tissue; at the apical foramen there is a small accumulation of inflammatory exudate cells, marking the center of irritation and inflammation. The portion of the root surface located within the granuloma shows resorption of both dentin and cementum, a common occurrence in periapical inflammatory processes of long standing.



FIG. 155.—Mesio-distal section through a lower first molar with broken-down crown and decomposed pulp. The radiograph of the specimen, taken before decalcification, is inserted in the lower right corner. It shows a considerable amount of bone destruction around the distal root end and a small amount of bone destruction around the mesial root end. This radiographic diagnosis is verified by the section, which shows a corresponding amount of bone destruction and granulation tissue (*GT*) at both root ends. *PM*, normal periodontal membrane in the upper portion of the root.

In Figure 155 is reproduced a mesio-distal section through a lower first molar with decomposed pulp and empty root canals. The radiograph shows a sharply outlined, radiolucent area around the mesial root end and a similar, smaller and more indefinite area at the apex of the distal root. The root canals appear empty. The histological section corroborates the radiographic findings. Around both root ends the otherwise regular and normal periodontal space is considerably wider; the space between root surface and bone is occupied by granulation tissue. In the periphery of the granuloma the tissue is fibrous; further toward the apex there is an increasing

number of polyblasts, which are densest in the tissue directly opposite the apical foramen.

Figure 156 shows a mesio-distal section through the gangrenous root of an upper first bicuspid. In the radiograph large, diffuse areas of bone destruction can be seen around the root ends of both bicuspids; in the histological specimen this area is filled with granulation

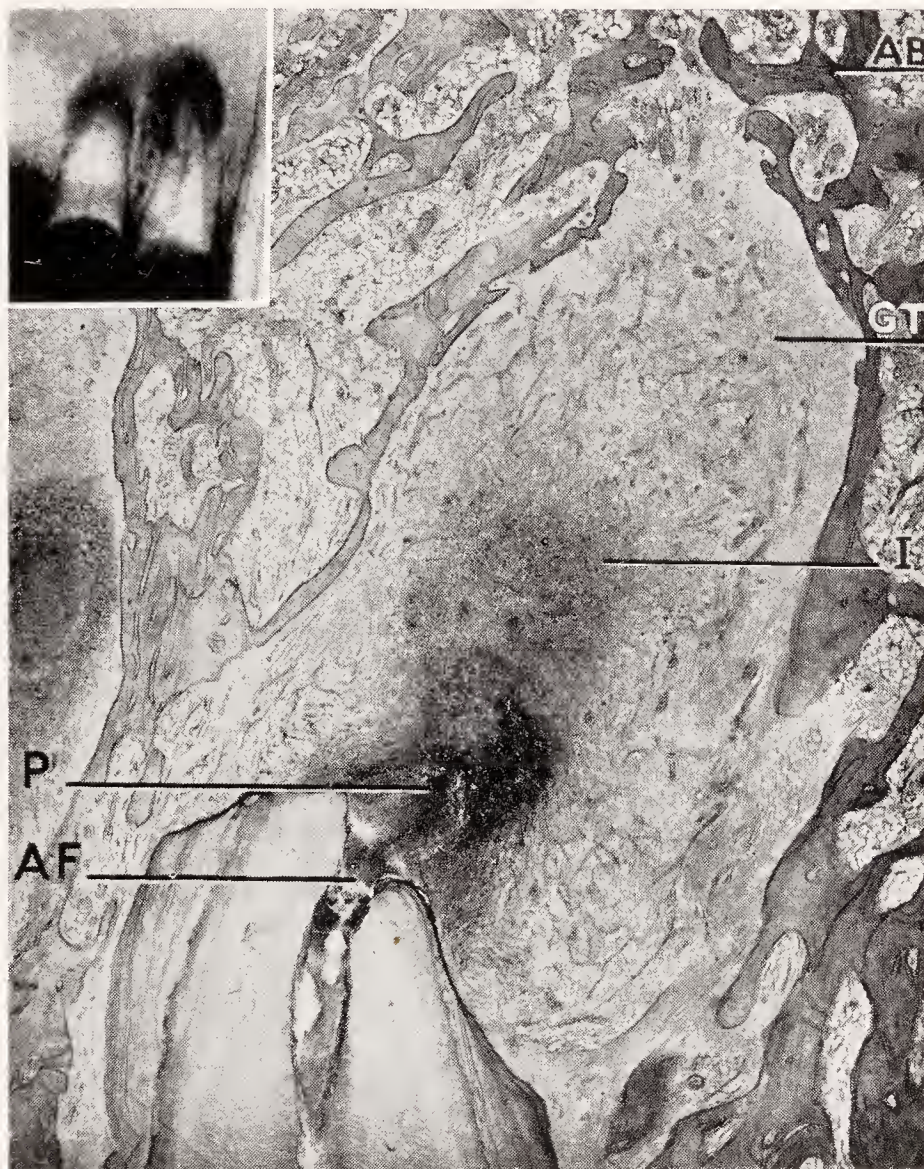


FIG. 156.—Mesio-distal section through the apex of an upper first bicuspid with granuloma. The radiograph of the specimen is inserted in the upper left corner. It shows large areas of bone destruction around the root ends of both upper bicuspids. *AF*, apical foramen; *P*, breaking down of tissue and formation of pus at the foramen; *I*, dense cellular infiltration next to the foramen; *GT*, granulation tissue; *AB*, alveolar bone.

tissue. In the periphery of the granuloma, next to the bone, the tissue consists principally of fibroblasts and connective tissue fibers with only a few inflammatory round cells. The fibrous connective tissue is arranged in the form of a capsule around the entire inflamed area. Toward the center of the granuloma the polyblasts increase in number. Opposite the foramen, the granulation tissue is densely infiltrated and is breaking down, discharging tissue debris and pus into the empty root canal.

The relationship between granuloma and surrounding alveolar bone is illustrated in Figure 157, which shows a granuloma at the apex of an upper central incisor with an imperfect root canal filling. In the radiograph a diffuse round area of bone destruction is visible. This bone cavity contains granulation tissue and a small area of liquefaction and pus formation opposite the apical foramen. In the

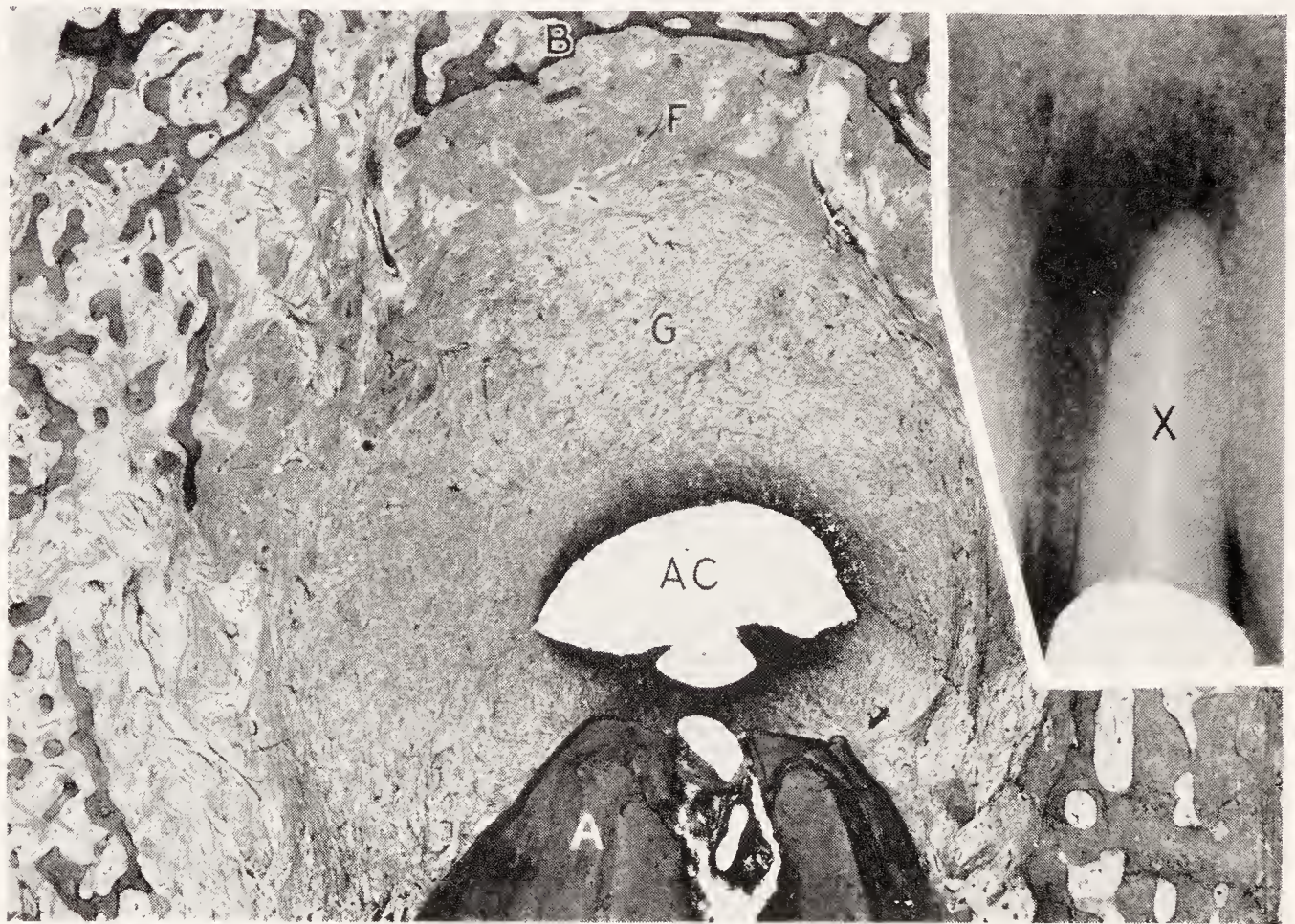


FIG. 157.—Granuloma with central liquefaction. No epithelium is present. The radiograph shows a diffuse area of bone destruction above the apex of an upper central incisor. Age, fifty-two years. *A*, apex; *AC*, abscess cavity containing pus cells; *G*, granulation tissue; *F*, fibrous tissue; *B*, bone trabeculae. (Kronfeld, Australian Dental Congress.)

periphery of the granuloma new bone trabeculae are being formed, which are growing from the outer bone wall into the granulation tissue. Such bone regeneration associated with apical inflammatory processes is not uncommon. It is an expression of the fluctuating character of the process: periods of activity during which the bone is resorbed alternate with periods of inactivity during which the bone is regenerated (Fig. 158). These periods of bone repair may be due to a decreased bacterial activity in the granuloma, to an increase in the defensive power of the body, or to drainage of the purulent exudate. If the purulent exudate in an apical abscess can drain through the root canal or through a sinus, bone forms along the

walls of the abscess cavity; if this drainage is blocked, however, bone formation is discontinued and resorption takes place instead.

The granulomas shown in Figures 154, 155, and 156 have the following characteristics in common: A distinct area of bone destruction around the apex is visible in the radiograph. This area, upon microscopic examination, is found to be filled with granulation tissue. The condition of the granulation tissue varies according to the intensity of the inflammation: in the periphery there is fibrous

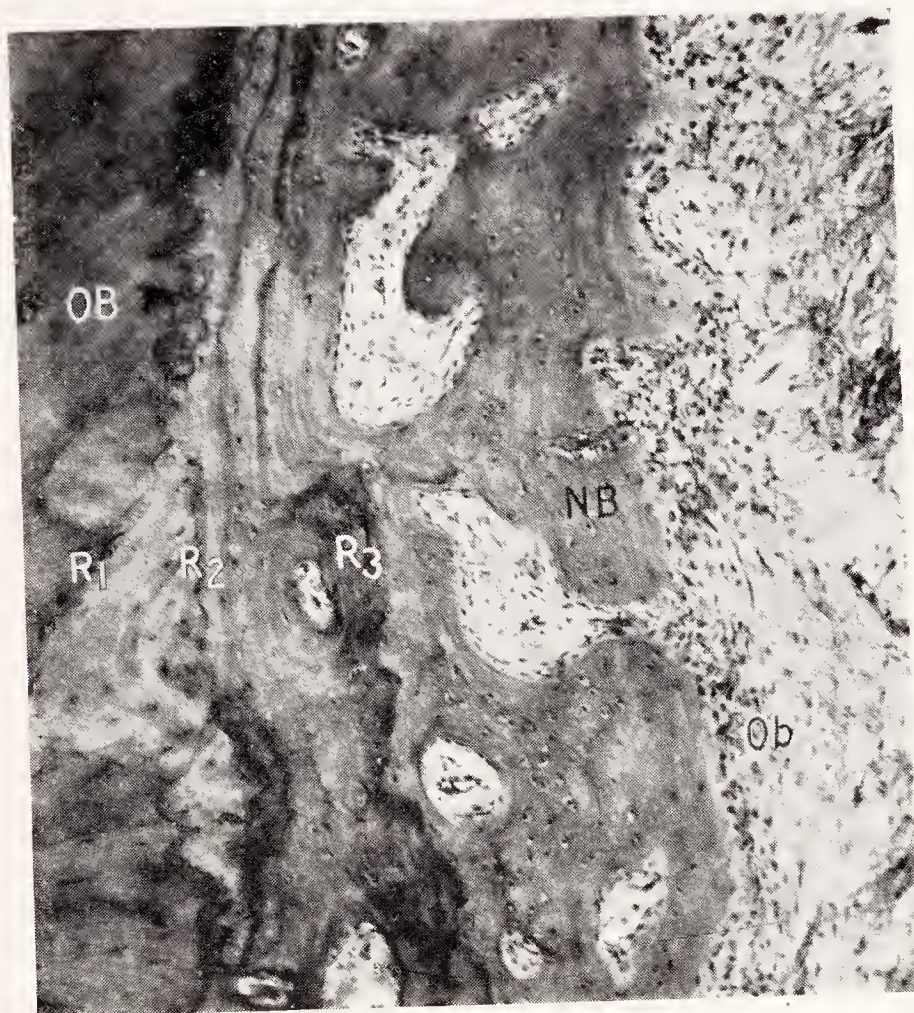


FIG. 158.—High magnification of the bone in Figure 157. *OB*, old bone; *R*₁, *R*₂, *R*₃, cementing lines indicating the extent of three alternating phases of bone resorption and deposition; *NB*, trabeculae of new bone; *Ob*, osteoblasts. (Kronfeld, Australian Dental Congress.)

connective tissue with only a few exudate cells; toward the apex the number of inflammatory exudate cells and capillaries increases, and at the apical foramen the tissue breaks down and pus is formed. The bone surrounding the granulation tissue is inactive, indicating that the inflammatory process is of a chronic nature and has no tendency to spread or involve additional bone.

Granuloma With Abscess Cavity.—Breaking down of tissue in the center of a granuloma leads to the formation of abscess cavities. They are distinguished from dental cysts by the absence of an epithelial lining. The wall of the cavity is formed by granulation tissue; this inflammatory lining is called a pyogenic membrane. Pus cells

and cell débris are continually being discharged from the walls into the cavity.

Figure 159 illustrates a mesio-distal section through an upper first and second bicuspid. The radiograph showed a diffuse, indistinct area of bone destruction around the root ends of these teeth. The microscopic examination reveals that only the first bicuspid has a decomposed pulp, the pulp of the second bicuspid being intact



FIG. 159.—Granuloma with central abscess cavity. Mesio-distal section through an upper first and second bicuspid. The second bicuspid is intact and vital; the first bicuspid has a decomposed pulp and an infected root canal. A granuloma with central abscess cavity has developed at the root end of this tooth. *AB*, alveolar bone; *CT*, connective tissue capsule; *P*, purulent exudate in the abscess cavity; *PM*, normal periodontal membrane in the lower portion of the root.

and vital. The root end of the first bicuspid projects into an abscess cavity about 3 mm. in diameter, which is partly filled with pus. The lining of the cavity consists of granulation tissue; farther out toward its periphery the character of the tissue approaches that of fibrous scar tissue, until, next to the bone, a plain connective tissue capsule is found. The surrounding bone shows evidence of resorption in some places.

Granuloma Connected With the Surface of the Alveolar Process by a Sinus.—Frequently the inflammation does not stay within the limits of the jaw bone, but the pus burrows through the bone and

the overlying mucoperiosteum to the surface of the jaw. The resultant opening is connected with the granuloma inside of the jaw by a duct lined with granulation tissue through which pus or serum is discharged. Such a condition is known as a sinus.

The development of a sinus may follow the perforation of an acute dento-alveolar process. After the acute symptoms have subsided, the perforation fails to heal, and a small, fistulous opening surrounded by granulation tissue remains, from which a yellowish exudate is discharged. Other times the sinus appears without any preceding acute symptoms, when a chronic inflammatory process around the



FIG. 160.—Sinus discharging upon chin. (Prinz.)

root end of an infected pulpless tooth gradually reaches the jaw surface and perforates the overlying tissues.

The location of the opening of a sinus depends upon the topography of the infected root end and its surrounding tissues. Most sinuses open into the labial or buccal vestibule. Occasionally they are found on the lingual surface of the upper or lower jaw, in the floor of the nose, and in the maxillary sinus. The inflammation may also perforate through the skin of the face. If the infected tooth is an upper cuspid the sinus may appear on the skin near the inner angle of the eye. Sinuses originating from lower incisors may perforate the skin of the chin (Fig. 160). Lower molars with decomposed, infected pulps have a tendency to form sinuses draining through the skin at the lower border of the mandible. Later, when such sinuses heal, they leave depressed, funnel-shaped scars.

Figure 161 shows a labio-lingual section through an upper central incisor with a decomposed, infected pulp. The apex is surrounded by granulation tissue. A sinus opens into the vestibule on the labial surface of the alveolar process, and pus is discharged through it into the oral vestibule.

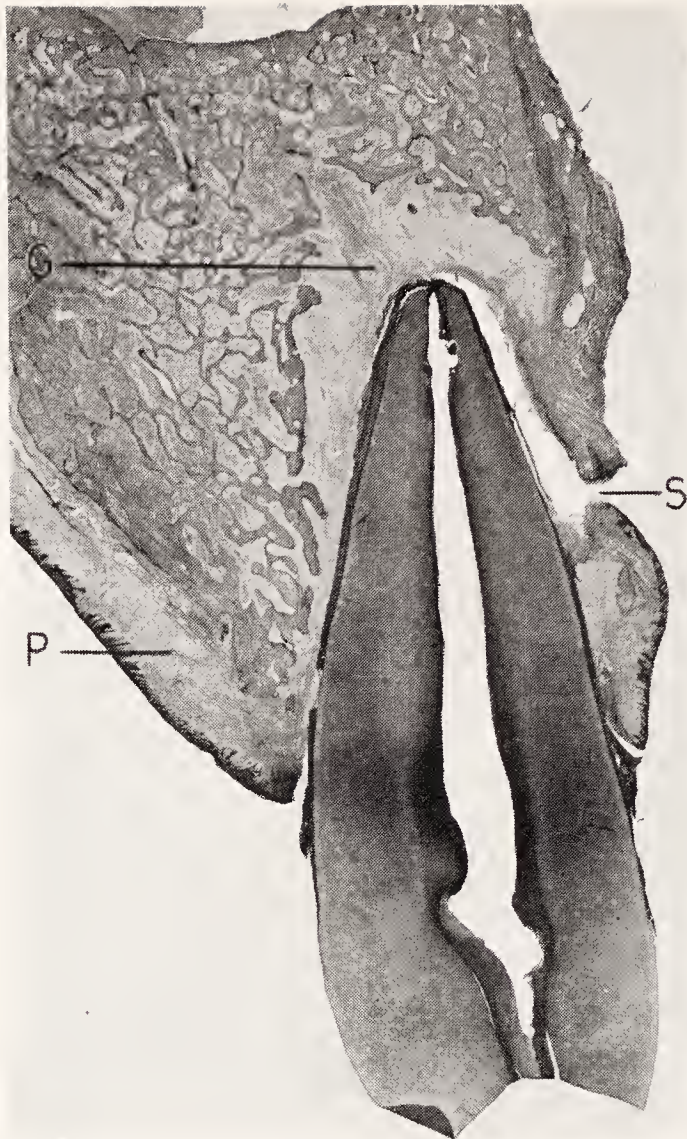


FIG. 161.—Chronic periodontitis with formation of a sinus. Labio-lingual section through an upper central incisor with decomposed pulp. *G*, granulation tissue surrounding the apex; *S*, sinus opening into the labial vestibule; *P*, fibrous tissue of the palate.

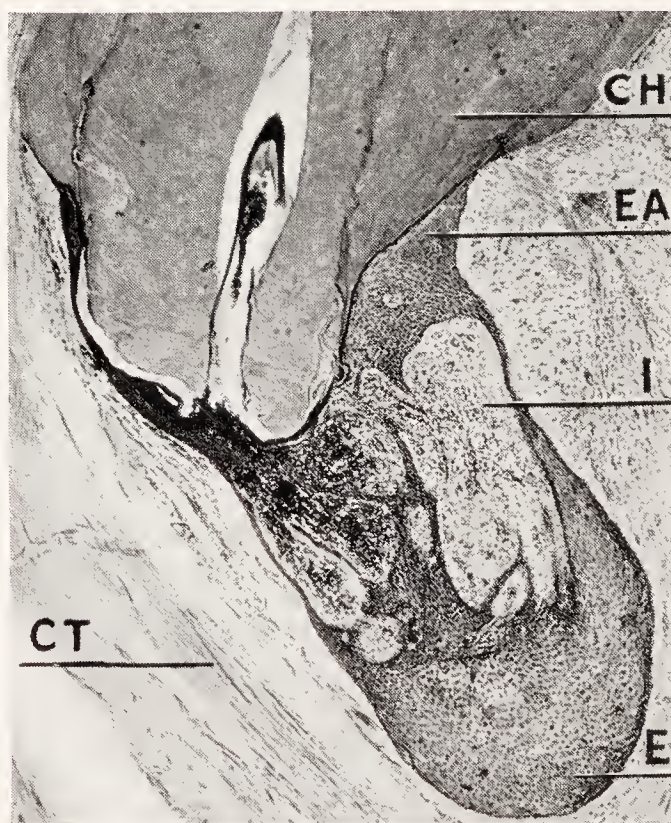


FIG. 162.—Root end of an upper bicuspid with decomposed pulp. Solid mass of epithelium present in the apical granuloma. *E*, epithelial proliferations at and near the root end; *EA*, epithelial attachment to the root surface; *CH*, cementum hyperplasia above the epithelial attachment; *I*, round cell infiltration in the connective tissue between the epithelial strands; *CT*, connective tissue capsule. (Gottlieb, Jour. Am. Dent. Assn.)

After the periapical inflammation has subsided, either because of extraction of the infected tooth or successful root canal treatment, the sinus disappears and the opening in the tissues heals.

Granuloma Containing Proliferated Epithelium.—Figure 162 shows a section through the root end of an upper bicuspid with decomposed pulp. Extensive epithelial proliferations are present at the root end.



FIG. 163.—Granuloma with marked fibrous incapsulation. The radiograph shows a sharply circumscribed, punched-out defect in the otherwise very dense bone. A large amount of epithelium is present, which has a tendency to grow around the granulation tissue. Lower lateral incisor; aged forty-five years. *G*, granulation tissue; *E*, epithelium; *F*, fibrous capsule surrounding the granuloma; *CB*, compact bone plate; *PM*, normal periodontal membrane. (Kronfeld, Australian Dental Congress.)

The epithelium is attached to the root surface and is surrounded by granulation tissue showing moderate round-cell infiltration. The squamous epithelium has formed a cuticle on the surface of the root, which is comparable to the secondary cuticle on the tooth surface beneath the epithelial attachment. Above the end of the epithelial attachment a cementum hyperplasia has developed.

An epithelium-containing granuloma at the apex of a broken-down lower central incisor is shown in Figure 163. The radiograph

shows a sharply circumscribed, punched-out bone defect. The microscopic picture reveals an area of granulation tissue with masses of proliferated epithelium, surrounded by a dense capsule of fibrous connective tissue that is attached to the cementum of the root. On the bone surface there is neither resorption nor apposition, indicating that the inflammatory process was stationary at the time of death. Serial sections reveal no evidence of a communication with the surface epithelium. This specimen is characteristic of a large group of similar granulomas sectioned *in situ*; all of them contain epithelium, and in all of them the bone between root and surface epithelium is undisturbed, compact bone without indication of a perforation or communication with the epithelium of the oral cavity.

Origin and Significance of Epithelium in Chronic Periodontitis.—All investigators who have studied the apical region of infected pulpless teeth have found epithelium in a large percentage of cases. Weber, for instance, reported that 49 out of 112 granulomas on extracted teeth contained epithelium. Stein examined 54 granulomas sectioned *in situ* and found epithelium in 35 of them. In Tsuzuki's material of 50 granulomas, epithelium was found in 21.

The percentage varies according to the technique used, as pointed out by Hill, who found epithelium in only about one-third of the granulomas of which merely a few sections were available, but in all granulomas of which serial sections had been mounted. This indicates that the probability of discovering small areas of epithelium in a granuloma is in direct ratio to the number of histological sections examined. Freeman reported epithelium in about one-half of over 200 granulomas. In the material that the author has examined, which consists of 68 infected pulpless teeth sectioned *in situ*, epithelium was found in 41, or 60 per cent; this percentage is about the same as Stein's, who examined the same type of material. It is possible that if serial sections were available through every granuloma, at least some epithelium would be found in each. For such an examination, root end and granuloma would have to be sectioned together, since small epithelial masses often lie close to the root surface and could easily be overlooked if the granulation tissue were detached from the root and sectioned alone.

The origin of epithelial tissue in apical inflammatory processes has been the subject of much controversy. The presence of small clusters and strands of epithelial cells in the periodontal membrane of man and animals has been known since 1885, when Malassez described and illustrated them. Then already Malassez realized the significance of these epithelial structures in the genesis of epithelium-

containing granulomas and root cysts. Subsequently many investigators studied the pathological and clinical significance of the epithelial proliferations derived from Malassez's epithelial rests. In American dental literature this problem was first systematically dealt with by Henrici and Hartzell, and Thoma.

In 1906 Grawitz expressed the opinion that the epithelium in granulomas and cysts originates from the oral mucous membrane and reaches the inside of the jaw through fistulous openings. His report brought forth much controversy and discussion; in 1932 James and Counsell stated: "The epithelium associated with chronic infections of the apical tissues of teeth has its origin in oral epithelium, and is always associated with a pre-existing sinus which may still be present or have become obliterated. . . . There is no reason to believe that the epithelial rests play any part in the production of the pathological epithelium."

Notwithstanding such occasional expressions to the contrary, most dental pathologists today consider the epithelial rests of the periodontal membrane as the usual source of the epithelium in granulomas and radicular cysts. The following facts support this view:

1. Very few granulomas and cysts communicate with the oral mucosa as compared to the number that are located inside the intact outer bone plate and have no such communication. Also, in most closed granulomas, the histopathological picture indicates that a sinus did not exist at some earlier time.

2. In the early stages of development of a granuloma the epithelial tissue is confined to the periodontal membrane in an area directly adjacent to the apical foramen. These early granulomas are completely incapsulated and surrounded by an intact layer of bone.

3. In man and animals the successive steps from the beginning of proliferation of the epithelial rests in the early stages of periapical inflammation to the typical epithelium-containing granulomas and cysts have been demonstrated (Feldmann, Hill, Hammer, Kronfeld). Actual transition from epithelial rests to proliferated epithelium has been found.

There is, however, no doubt but that occasionally secondary epithelization of an apical abscess cavity takes place in the manner described by Grawitz and others. But even in the cases in which a continuous mass of epithelium extends from the oral mucosa to a granuloma or cyst, it is impossible to decide whether this epithelium originally grew from the granuloma or cyst outward or from the mucous membrane inward. Only if the epithelium in a granuloma

or cyst were of a type not normally encountered in the jaw would the conclusion be warranted that this epithelium came from without the jaw. This is true in those cases in which ciliated columnar epithelium is found in the apical region of upper teeth. This epithelium is derived from the maxillary sinus and has proliferated into an apical abscess cavity or radicular cyst that communicated with the antrum. (See Fig. 174.)



FIG. 164.—Radicular cyst. (Prinz.)



FIG. 165.—Radicular cyst. (Prinz.)

Radicular cysts develop from epithelium-containing granulomas. It is impossible to draw a sharp dividing line between such granulomas and radicular cysts, since almost every epithelium-containing



FIG. 166.—Small radicular cyst formed by epithelization of the wall of an abscess cavity at the root end. Upper molar. *E*, epithelial lining of the cyst cavity; *P*, purulent exudate within the cyst next to the apical foramen; *EA*, epithelial attachment of the cyst epithelium to the root surface; *CT*, connective tissue; *AB*, alveolar bone; *R*, resorption of the alveolar bone. The presence of bone resorption indicates that the cyst was growing at the time of death.

granuloma contains areas that microscopically appear as the earliest stages of cyst formation. Therefore, granulomas containing only minute cysts will be described separately from those with definite epithelium-lined cyst cavities.

Radicular Cyst (Root Cyst).—The squamous epithelium at the root ends of infected pulpless teeth has a tendency to form cystic cavities. A cyst is a pathological cavity lined with epithelium and containing a fluid or semifluid. A cyst found at the root end of an infected pulpless tooth is called a radicular or root cyst. Its epithelial lining is derived from the epithelial rests of the periodontal membrane (Figs. 164 and 165).

Radicular cysts can originate in two ways: An abscess cavity may develop in the granulation tissue surrounding the infected apex, and the epithelium, because of its inherent tendency to grow over raw surfaces, covers the walls of this abscess cavity. The other possibility is cystic degeneration within the epithelial strands themselves; the epithelial masses are hollowed out, enlarged, and transformed into cysts.

Figure 166 shows a section through one of the roots of an upper second molar with decomposed pulp. There is a granuloma at the apex, in the center of which a small cystic cavity lined with epithelium (radicular cyst) has developed. The inflammation is rather active, as indicated by the presence of a large number of polyblasts in the granulation tissue and the accumulation of pus cells and cell débris around the apex. The epithelium of the cyst cavity is attached to the surface of the root with an epithelial attachment. The pus at the apical foramen is the result of a discharge of inflammatory cells, leukocytes and polyblasts, from the granulation tissue through the epithelial lining into the cyst cavity. Osteoclasts on the surrounding bone indicate that the cyst has a tendency to grow.

In large radicular cysts the inflammation is sometimes of a very low grade. The fibrous tissue surrounding them shows few inflammatory changes, and the cyst wall is smooth and lined with a thin, uniform layer of stratified squamous epithelium (Figs. 167 and 168). The contents of the cyst gradually change from a purulent exudate to a clear, amber fluid that sometimes contains many long, thin, spear-shaped crystals of cholesterol. Cholesterol is normally found in small quantities in the bile and blood. Under pathological conditions it is present in some types of gall stones (cholesterol stones), and it may also be found anywhere in the body where tissue decomposition, especially decomposition of epithelial tissue, takes place. Pure cholesterol is a solid, white substance that crystallizes in the

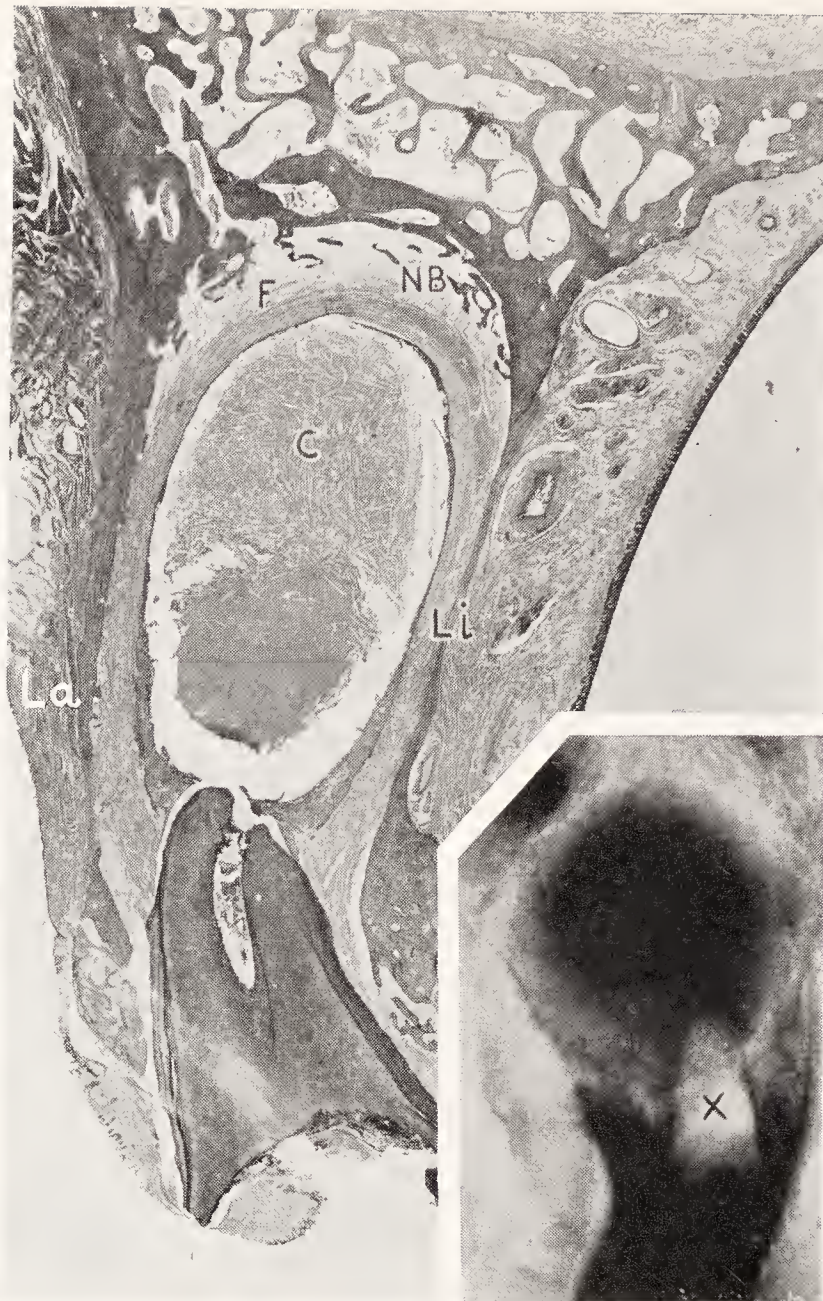


FIG. 167.—Radicular cyst. The radiograph shows a large round area of bone destruction. Upper cuspid; aged fifty-six years. *C*, cyst cavity containing cholesterol; *F*, fibrous capsule; *NB*, trabeculae of new bone; *La*, *Li*, thin labial and lingual bone plates. (Kronfeld, Australian Dental Congress.)



FIG. 168.—Higher magnification of cyst wall in Figure 167. *C*, cyst cavity; *E*, epithelial lining of cyst; *F*, fibrous capsule of cyst free of inflammatory infiltration, *B*, trabeculae of new bone. (Kronfeld, Australian Dental Congress.)

form of long, thin needles; it is soft and has a silky, oily feeling. Chemically it is a monatomic alcohol derived from a complicated hydrocarbon.

Cholesterol crystals are frequently observed suspended in the fluid of dental cysts, appearing as fine, shiny flakes. They seem to be the end-product of the disintegration of epithelial cells from the cyst wall. If specimens of cysts are treated with alcohol or ether during the preparation of microscopic slides, the cholesterol is dissolved; however, in the sections, the spaces originally occupied by the needle-shaped crystals can be recognized easily (cholesterol slits) (Fig. 169).

Radicular cysts in the jaw occasionally become very large and then cause a slowly growing prominence on the outer surface of the



FIG. 169.—Cholesterol slits (CS) in a radicular cyst. The cholesterol forms needle- or spear-shaped crystals, which leave slits of the same shape after the cholesterol has been dissolved in the preparation of the specimen.

jaw. At first this prominence is covered by a thin plate of bone, which later disappears as a result of the continued pressure of the cyst. Then the cyst wall lies directly beneath the oral mucosa and can be felt by palpation.

A large dental cyst of this type is illustrated in Figure 170. The patient, a boy, aged eight years, had a slowly growing, painless swelling on the right side of the maxilla. The right deciduous central and lateral incisors were still in place; on the left side the permanent central and lateral incisors had already erupted. Both right deciduous incisors appeared clinically intact; the deciduous central incisor, however, was discolored, which, in connection with the history of a trauma (blow), suggested death of the pulp with subsequent periapical infection. The radiographic examination revealed the presence of a large cavity in the bone involving the entire anterior portion of the right maxilla. In the upper part of this cavity the displaced crowns of the permanent incisors are visible. The root

end of the deciduous central incisor is resorbed. These radiographic findings lead to the following diagnosis: traumatic death of the pulp followed by infection and inflammation of the periapical tissue and resorption of the root; development of a radicular cyst with displacement of the crowns of the permanent teeth by the pressure of the growing cyst sac.



FIG. 170.—Large radicular cyst originating from a deciduous right upper central incisor following traumatic necrosis of the pulp. The radiograph shows the resorbed root end of the deciduous incisor projecting into the cyst cavity. This observation is substantiated by the microscopic finding of extensive resorption of the root end. *NP*, necrotic pulp tissue; *Cy*, cyst cavity; *E*, epithelial lining of the cyst; *CT*, connective tissue capsule of the cyst; *EA'*, epithelial attachment of the oral epithelium to the enamel; *EA''*, epithelial attachment of the cyst epithelium to the resorbed root surface.

This diagnosis was verified by the operation, which consisted of the removal of the central and lateral deciduous incisors together with the surrounding soft tissues and part of the cyst sac. In the months following the operation the misplaced permanent incisors began to erupt and finally assumed their normal place in the jaw.

The specimen removed during the operation was sectioned in labio-lingual direction. Figure 170 shows the central incisor with its intact crown and necrotic, decomposed pulp. The upper half of the root has been extensively resorbed; the root end extends into a large cystic cavity which, through the apical foramen, communicates with the contents of the pulp canal. The wall of the cyst consists of a dense, fibrous connective tissue capsule with a lining of stratified squamous epithelium.

Classification of Radicular Cysts.—Radicular cysts can be classified according to the degree of differentiation and maturity of cyst epithelium and surrounding connective tissue (Tsuzuki). In the early stages of cyst formation the epithelium is irregularly proliferated; there is dense cellular infiltration of the surrounding loose connective tissue. In the later stages the epithelium is smooth and regularly arranged, resembling the epithelium of the normal oral mucosa; the surrounding connective tissue is fibrous, with very little or no cellular infiltration. This mature cyst epithelium, however, may later undergo secondary proliferation and inflammatory changes if the cyst becomes acutely infected.

The following is a tentative division of radicular cysts:

Growing young cyst: Immature and incomplete epithelial lining; inflammatory exudate cells in epithelium and connective tissue; no definite fibrous capsule; bone resorption. Radiographically, diffuse bony outline.

Mature cyst, stationary or only very slowly growing: No or few inflammatory exudate cells in epithelium and connective tissue; fibrous capsule; compact layer of aplastic bone surrounding the cyst. Radiographically, definite bony plate surrounding the bone defect.

Infected cyst: Epithelium partly destroyed; cyst wall formed by granulation tissue; dense cellular infiltration of epithelium and connective tissue; bone resorption. Radiographically, at the onset, definite bone plate; later, diffuse bony outline.

Mature cyst, decreasing in size: No or few inflammatory exudate cells; fibrous capsule; new bone growing from the bone wall. Radiographically, diffuse bony outline.

COMPARISON OF THE RADIOGRAPHIC AND MICROSCOPIC PICTURE IN CHRONIC PERIAPICAL INFECTION.

A comparison of the radiographs in chronic periodontitis with the corresponding tissue sections indicates that, with the exception of very advanced stages, it is impossible to distinguish radiographically between solid granuloma, apical abscess, and radicular cyst.

A sharply outlined bony defect, like the one in Figure 163, denotes a stationary inflammatory process surrounded by dense bone; whether the condition is a cyst or not cannot be told from the radiograph. If the surrounding bone is thin and cancellous, and especially if the periapical inflammation is progressive, or if there is formation of new bone along the walls of the bone cavity, the outline of the bone appears diffuse and indefinite (Fig. 157). If the bone destruction is very extensive the diagnosis of a cyst is probable, but never absolutely certain. It cannot be overemphasized that radiographs do *not* show infections, granulomas, or cysts, but merely differences in the relative density of the bone shadow.

ACUTE EXACERBATION OF CHRONIC PERIAPICAL INFLAMMATION.

Most acute dento-alveolar abscesses develop from preëxisting chronic inflammations, such as granulomas or cysts. The reasons for such an acute exacerbation are manifold. Sometimes a chronic periodontitis may turn into an acute abscess for no apparent reason; perhaps the virulence of the bacteria increased or the resistance of the body decreased to a point where the wall of defense that had been built up around the chronic process broke down. Frequently, however, some outer interference is responsible, especially a change in drainage conditions. For instance, if a chronic abscess discharges its exudate through an open root canal into the oral cavity, usually no clinical symptoms are present. If, however, the root canal becomes obstructed by coagulum or débris, the retention of exudate may cause an acute exacerbation with pain, swelling and fever, increase in the number of leukocytes, and acute abscess formation. Acute exacerbations following root canal therapy on pulpless teeth may be explained in the same way. The manipulations in the root canal disturb the equilibrium between the bacteria and the defense mechanism of the body; the infection spreads and causes an acute abscess.

Figure 171 shows a mesio-distal section through the posterior part of an upper jaw. The crowns of the upper second and third molars are broken down and the pulps are decomposed. An acute alveolar abscess around the roots of these teeth has developed from a chronic inflammation. Around the apices there is a cavity of irregular outline and of more than 1.5 cm. in diameter which is filled with pus; its walls are densely infiltrated with inflammatory round cells. The infiltration extends into the marrow spaces of the surrounding



FIG. 171.—Exacerbation of chronic periapical inflammation and formation of an acute abscess. Mesio-distal section through the posterior portion of the maxilla. Second and third molar with decomposed infected pulps. The radiograph in the lower left corner shows the carious destruction of the crowns and the extensive periapical bone destruction. *A*, abscess cavity extending over the lingual roots of both molars; *MS*, floor of the maxillary sinus; *Hy*, hyperplastic gum tissue located in the cavity on the distal side of the second molar (gingival polyp).

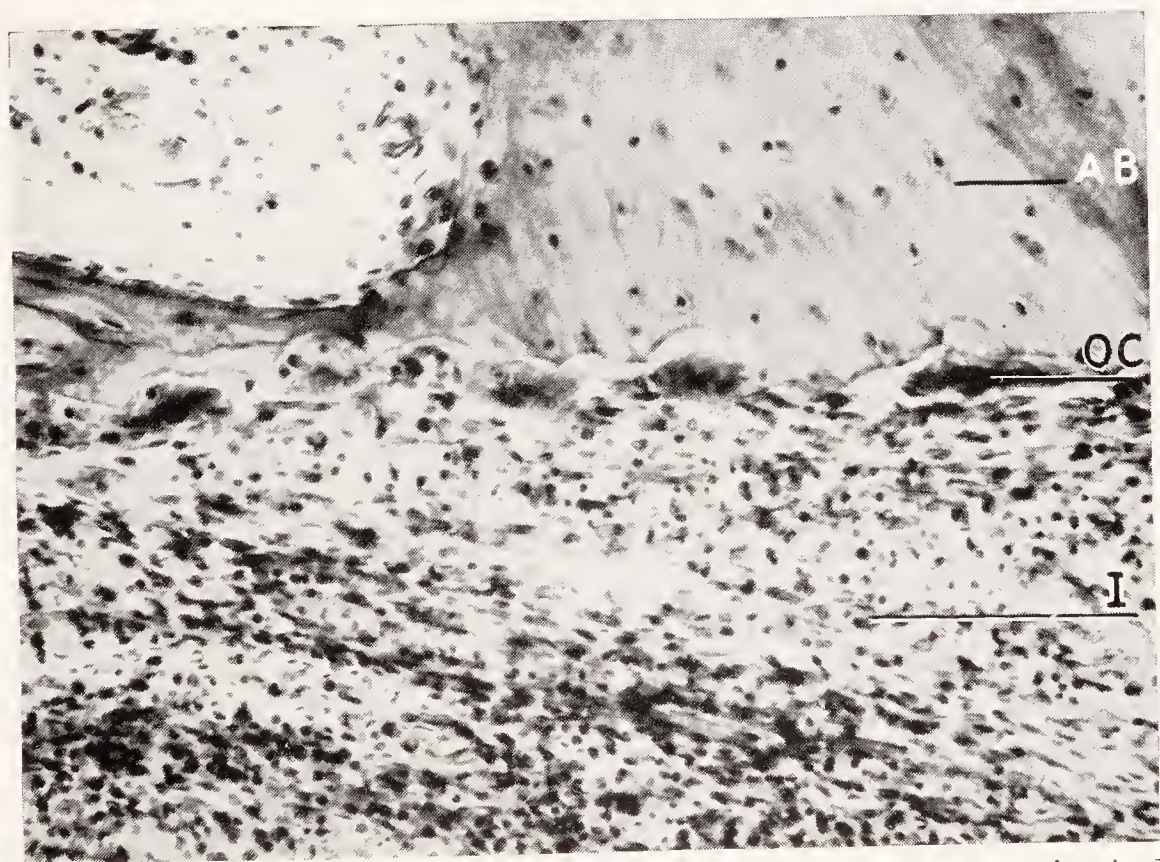


FIG. 172.—Higher magnification of the periphery of the abscess cavity in Figure 171. *AB*, alveolar bone; *OC*, osteoclasts; *I*, inflammatory exudate cells, mostly polymorphonuclear leukocytes. The presence of these leukocytes and of the large number of osteoclasts indicates an acute inflammatory process with progressive bone destruction.

bone. The presence of a fibrous capsule and of epithelium in the granulation tissue around the abscess cavity indicates that the inflammation was of long standing and that the acute exacerbation had developed only shortly before death. A higher magnification of the periphery of the abscess cavity reveals the spreading of the inflammation beyond the fibrous capsule; the neighboring bone is lined with osteoclasts, indicating active bone resorption (Fig. 172). Almost all of the inflammatory round cells in the tissue around the abscess and in the pus are polymorphonuclear leukocytes.

PERIAPICAL INFLAMMATION IN ITS RELATIONSHIP TO THE MAXILLARY SINUS.

The root ends of the upper posterior teeth are often in close contact with the maxillary sinus; thus, inflammatory processes originating from these teeth may influence the condition of the sinus.

The anatomy of the maxillary sinus has been discussed in the preceding chapter. A few specimens of chronic periapical inflammation in relation to the floor of the antrum will be illustrated here.

Figure 173 shows a mesio-distal section through the lingual root of an upper second molar with decomposed pulp. The root projects above the floor of the maxillary sinus, its apex lying about 4 mm. higher than the deepest point of the sinus. The root is surrounded by a thin plate of bone, which forms both the wall of the sinus and the alveolar bone of the tooth. At the apex a granuloma has developed; as a result, the distance between the root surface and the bone is greater than the thickness of the normal periodontal membrane on the sides of the root. Originally the root was surrounded by a uniformly thick periodontal membrane and a normal plate of bone; later, when the inflammation developed, the bone was resorbed on the inside of the alveolus, resulting in compensatory formation of bone on the outer side of the alveolar plate. The mucosa of the maxillary sinus covering the prominent root is histologically normal. If, however, such a chronic periapical inflammation persists over a long period of time, the tissues between the granuloma and the antrum may eventually be destroyed and the latter may become involved. This condition is illustrated in the next specimen, Figure 174, a mesio-distal section through the posterior part of the upper jaw. It shows an upper first and second molar with broken-down crowns and decomposed pulps and a third molar with intact pulp. The maxillary sinus is large, extending over the root ends of all three molars. At the root end of the second molar, a cyst of about 1 cm. in diameter has developed; the apex of the tooth projects above the

floor of the cyst. There is a perforation in the top of the cyst through which the cyst cavity communicates with the antrum. The cyst is partly filled with desquamated epithelial cells, polyblasts, and coagulated fibrin. In its lower portion, the wall of the cyst is

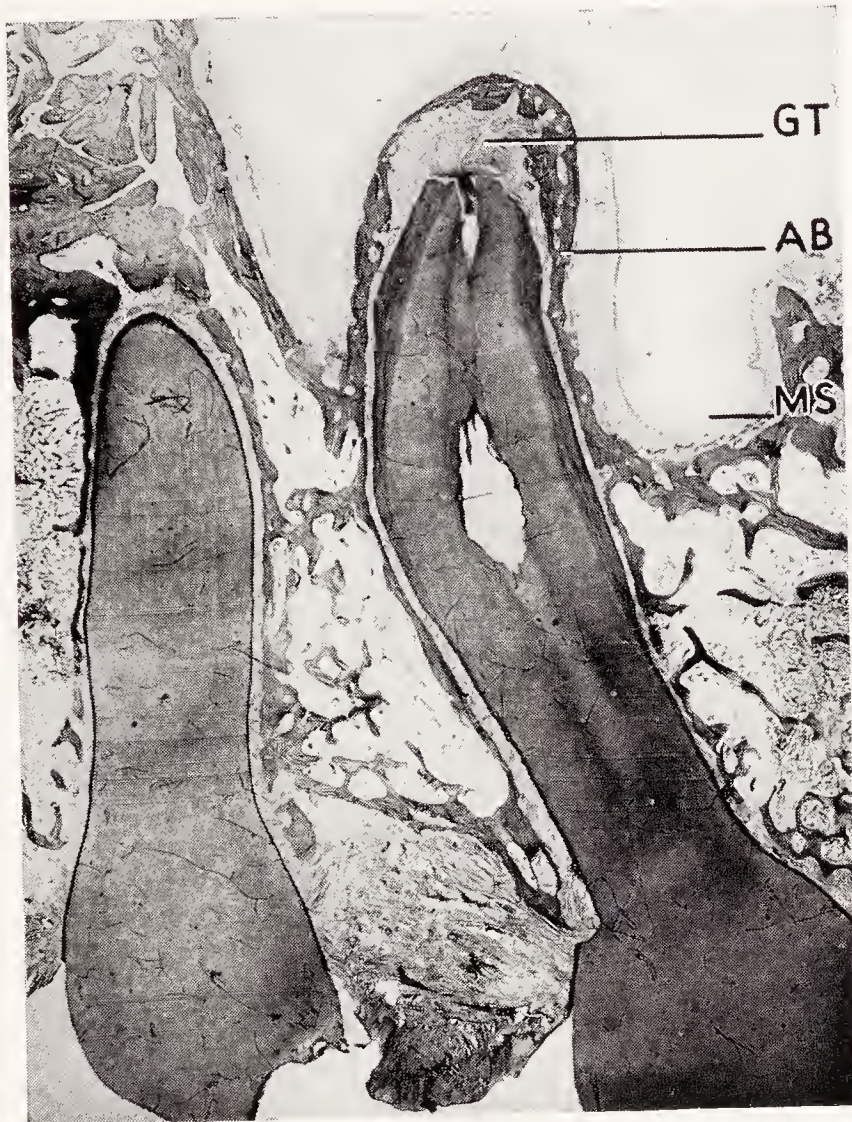


FIG. 173.—Relationship between periapical infection and maxillary sinus. Mesio-distal section through the lingual root of an upper second molar with decomposed, infected pulp. The apical foramen of this root is located about 4 mm. above the deepest point of the maxillary sinus, *MS*. *GT*, granulation tissue at the root end; *AB*, alveolar bone prominent over the floor of the maxillary sinus. The mucosa of the maxillary sinus is intact.

lined with stratified squamous epithelium (Fig. 175) like that usually found in radicular cysts. In its upper part, however, the epithelial lining consists of ciliated columnar epithelium (Fig. 176), which is characteristic of the accessory sinuses of the nasal cavity. Evidently the lower portion of the cyst still contains the original cyst epithelium, whereas the upper part is covered with epithelium originating from the adjacent maxillary sinus. The mucosa of the maxillary sinus covering the cyst shows extensive inflammatory changes, indicating the presence of a circumscribed sinusitis. The rest of the mucosa is normal. The extraction of the second molar would, at least temporarily, create a communication between oral cavity and maxillary sinus.



FIG. 174.—Relationship between periapical infection and maxillary sinus. Mesio-distal section through the posterior part of the maxilla with first, second, and third molars. All root ends are close to the floor of the maxillary sinus. The pulp of the second molar is decomposed and infected. At the root end a cyst has developed, which caused an elevation of bone over the floor of the maxillary sinus and finally perforated into the sinus at the highest point of this elevation. The lower portion of the cyst contains purulent exudate.

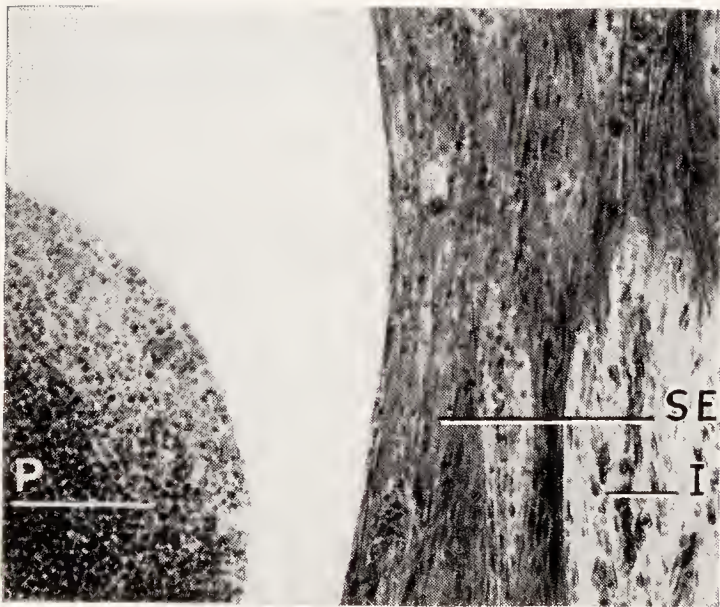


FIG. 175

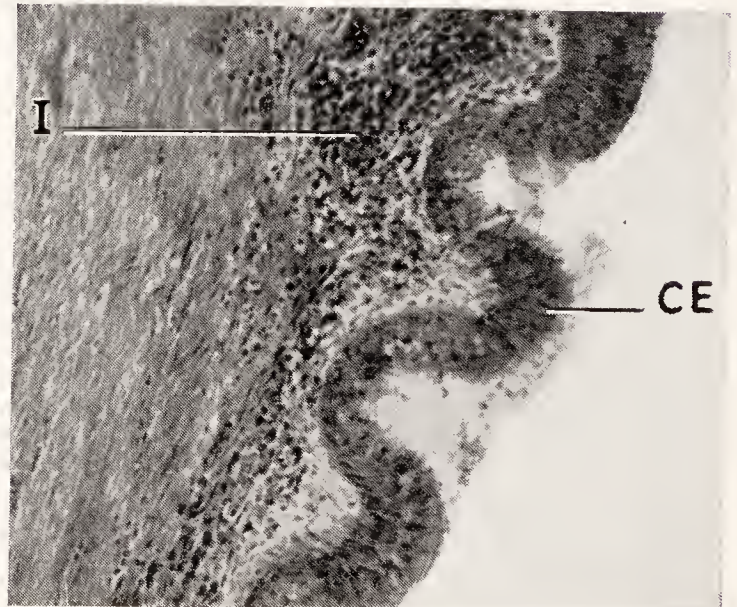


FIG. 176

FIGS. 175 and 176.—Higher magnification of the cyst wall in Figure 174.

FIG. 175.—Lower portion of the cyst wall. *SE*, stratified squamous epithelium; *I*, subepithelial round-cell infiltration; *P*, purulent exudate.

FIG. 176.—Upper portion of the cyst wall. *CE*, columnar epithelium originating from the maxillary sinus; *I*, subepithelial round-cell infiltration (polyblasts).

RESIDUAL INFECTION.

During the past decade clinicians and pathologists have paid much attention to what is commonly termed "residual infection." Judging from the microscopic examination of biopsy specimens removed from jaws, it would seem that most diagnoses of residual infection based on radiographic evidence alone are erroneous; rather,

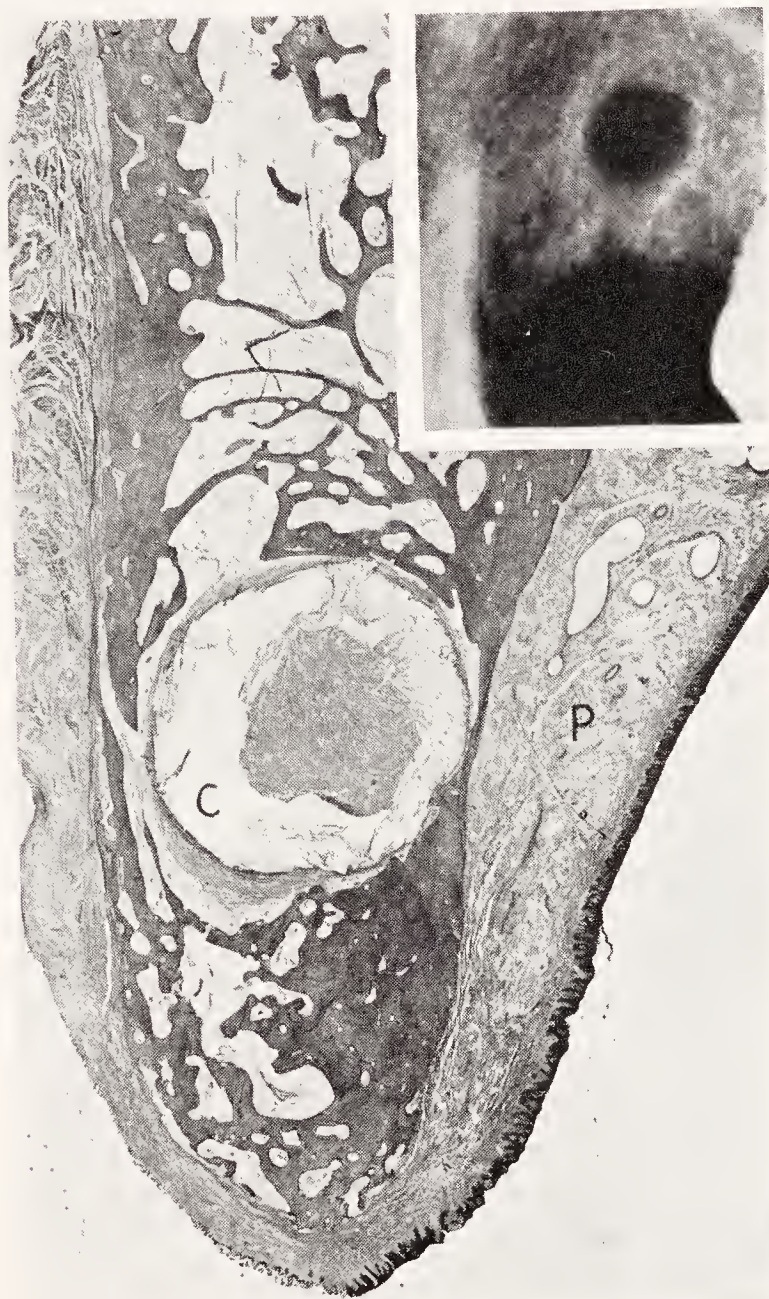


FIG. 177.—Small radicular cyst that remained in the jaw after loss of the teeth. The radiograph shows a distinct, round bone defect in an edentulous area. The corresponding tissue section reveals a radicular cyst without inflammatory tissue reaction either in the cyst or in its vicinity. Edentulous area of upper cuspid and first bicuspid; aged fifty-six years. *C*, cyst containing cholesterol; *P*, palatine tissue. (Kronfeld, Australian Dental Congress.)

the underlying pathological condition is fibrosis in an empty alveolus, bone sclerosis, or simply a marrow cavity. In the vast majority of granulomas and cysts, the infection terminates with the extraction of the infected tooth.

On rare occasions a radicular cyst may remain in the jaw following extraction of the tooth. Such a condition is shown in Figure 177.

The specimen was obtained from an edentulous, well-healed area of an upper jaw. The radiograph reveals a circular bone cavity with an indication of an alveolus leading toward the jaw ridge. The histological sections show a round cyst of 5 mm. in diameter, containing cholesterol. The bony walls surrounding it are aplastic. The cyst epithelium and the connective tissue of the cyst membrane are free of inflammatory changes, and the Gram-Weigert stain shows no microorganisms. Therefore, it seems preferable to refer to this condition as a residual cyst rather than a residual infection, since there is no actual evidence of infection.

CONDENSING OSTEITIS (BONE SCLEROSIS) CAUSED BY CHRONIC PERIAPICAL INFLAMMATION.

Chronic inflammations about the root ends of infected teeth do not always lead to bone destruction. Sometimes a low-grade irrita-



FIG. 178.—Bone sclerosis caused by chronic periapical infection. (Bunting.)

tion of long standing, instead of resorbing the bone, causes circumscribed bone hyperplasia, known as bone sclerosis.

Histologically, the sclerotic area may consist of denser bone trabeculae than those of the surrounding bone, in which case the border between normal and sclerotic bone is indistinct, or there may be an island of solid, compact bone that stands out clearly against the surrounding cancellous bone.

The radiograph shows a corresponding difference between these two types of condensing osteitis. Sometimes there is a diffuse area of denser bone around a tooth with chronic periapical infection; again, a sharply outlined, dense mass of bone may be located next to the diseased root end (Fig. 178).

No therapy is known for this condition. It is difficult to localize the condensed area of bone and to remove it surgically. The extraction of the tooth that caused the bone inflammation does not cause the area of bone condensation to disappear; it usually persists.

Such areas of dense bone have sometimes been erroneously diagnosed as areas of residual infection in the jaws.

Figure 179 shows a mesio-distal section through the mandible. A small root tip of a lower molar is attached superficially to the jaw ridge. The bone underneath it differs from that in the rest of the jaw; the trabeculae are much denser, with only very small marrow spaces between them. In this case the chronic irritation from the infected root tip was responsible for the condensing osteitis under the root. It is probable that this bone area would have remained in the jaw after the elimination of the root.

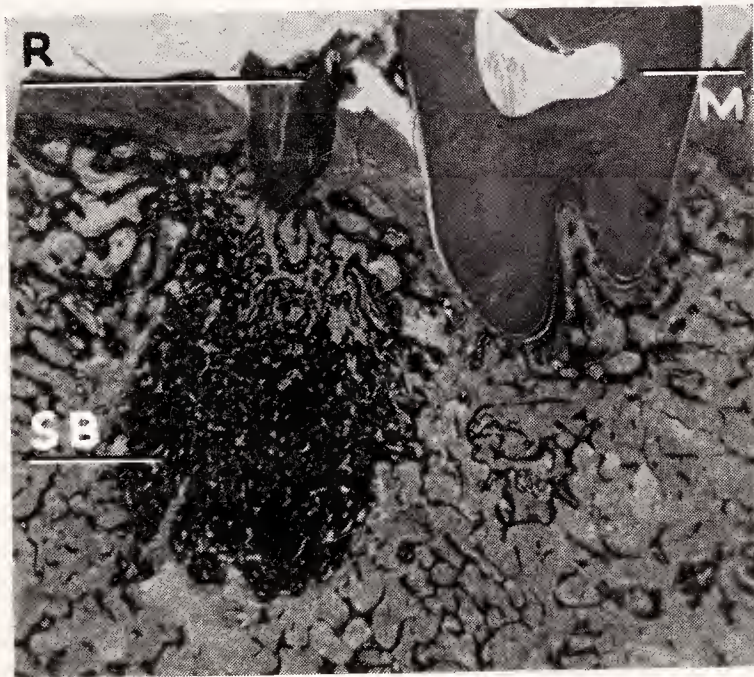


FIG. 179.—Bone sclerosis due to chronic low-grade inflammation around the distal root of a broken-down first molar. *R*, root with carious surface; *SB*, area of sclerosed bone below the root end; *M*, intact second molar.

In Figure 180 a radiograph of the right maxilla of a man, aged thirty-eight years, is reproduced. The jaw was edentulous in this area; the soft tissues on the ridge were completely healed. The radiograph shows a dense area that looks like a root tip in the alveolar ridge in the region of the molars. However, upon close examination no periodontal space could be found; there is a diffuse transition between the dense area and the surrounding cancellous bone. This makes the diagnosis of an area of condensed bone most probable. The histological specimen shows a sharply outlined area of compact bone in this region (Fig. 180). Neither the bone nor the surrounding fat marrow shows any sign of inflammation, and it is impossible at present to determine the etiology of the circumscribed bone condensation. Most probably, at some previous time an infected root caused irritation and bone condensation, similar to the condition illustrated in Figure 179. Later on, this root was lost and the dense

bone remained as a bony scar. It is also possible that originally a granuloma was present around an infected root and that, after the inflammation had subsided, the fibrous scar tissue was changed into compact bone.

In view of the microscopic findings in these and several other cases of condensing osteitis caused by infected teeth, surgical interference appears contraindicated in such conditions. After the extraction of the infected tooth, the remaining bony scar is free of inflam-

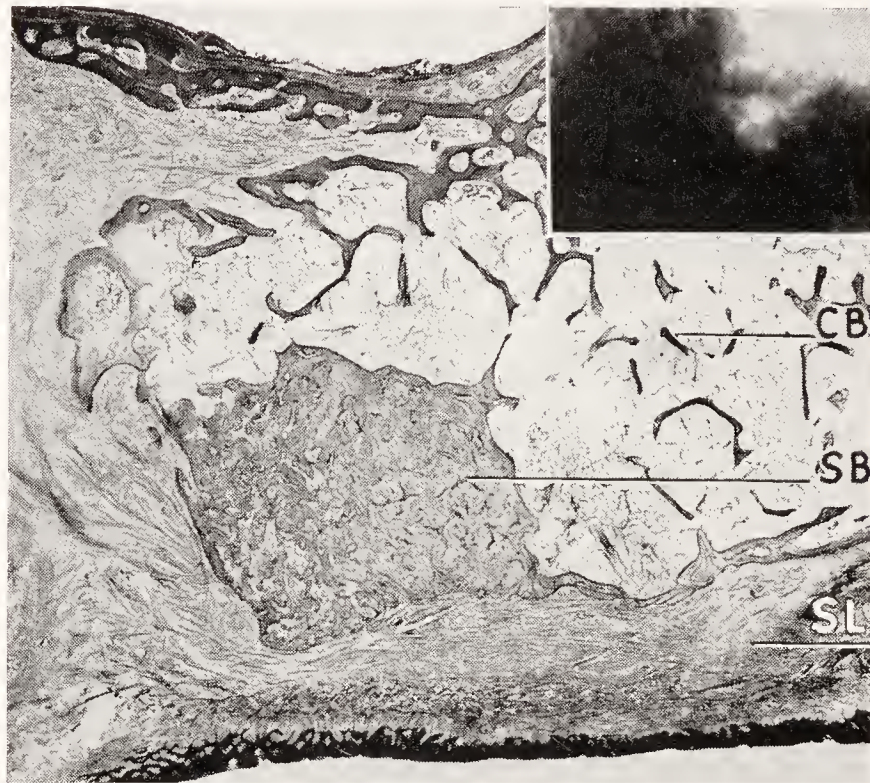


FIG. 180.—Mesio-distal section through an edentulous maxilla. The radiograph in the right upper corner shows a distinctly outlined radiopaque area in the posterior portion of the maxilla. *SL*, soft tissue lining of the edentulous maxilla; *CB*, cancellous bone of the maxilla; *SB*, area of compact bone lying next to the surface of the bone.

mation; besides, since the transition from condensed bone to normal cancellous bone is frequently indistinct, complete surgical removal of all sclerotic bone might be very difficult.

BACTERIOLOGY OF CHRONIC PERIODONTITIS.

The presence and distribution of pathogenic microorganisms in infected pulpless teeth, granulomas, and radicular cysts can be studied by cultural methods and by staining bacteria in the tissues. The first method, by which bacteria are taken from the infected field and grown in suitable culture media, gives the best results as far as the actual detection and identification of the microorganisms are concerned. The second method makes it possible to determine where these bacteria are located and how they are distributed in the teeth and their surrounding tissues.

All investigators agree that the root canals of infected pulpless teeth contain large masses of bacteria. Some of these are saprophytes that live on the decomposing matter in the canal; others are pathogenic, and among the latter the streptococci are the most important group. The presence of bacteria can easily be demonstrated in smears from the root canal of an untreated pulpless tooth.

It is not quite so simple to answer the question as to whether or not bacteria are present in granulomas and radicular cysts, for the findings vary with the bacteriological technique used. A few investigators have reported bacteria in all granulomas. The majority of recent observers, however, agree that granulomas and cysts are found sterile in quite a large percentage of cases. In Gram-stained sections through infected pulpless teeth *in situ* that were examined by the author, bacteria in abundance were always found within the root canal, but granulation tissue and cysts attached to the apices of these teeth were often free of microorganisms. Large radicular cysts of the mature type, with a well-developed epithelial lining, are usually sterile.

These observations are not difficult to interpret if the true significance of dental granulomas is understood. A granuloma is not an area in which bacteria *live*, but in which they are *destroyed*. The bacteria live and multiply in the infected root canal. As they grow they tend to pass through the apical foramen and invade the periodontal soft tissues. Opposite this zone of danger the body builds up a barrier of granulation tissue that destroys the bacteria as they grow out of the root canal and prevents them from entering the periodontal tissues. As a rule, this defense mechanism is satisfactory, as demonstrated by the relative scarcity of systemic infection of dental origin in comparison to the great abundance of localized chronic dental infections. Occasionally, for unknown reasons, this defense mechanism fails; then large quantities of bacteria may be found in the granuloma.

Harndt who examined many granulomas on extracted teeth by the staining method came to the following conclusions: Most simple granulomas are sterile. Bacteria are always found, however, in granulomas with acute inflammatory changes that have caused clinical symptoms of pain and an incipient acute dento-alveolar abscess. There are no bacteria in radicular cysts, unless they happen to be acutely inflamed at the time of examination. The main source of bacteria is always the untreated or poorly treated root canal, but not the tissue outside the root end.

The relationship between bacteria in an infected root canal and the periapical granulation tissue lends itself exceptionally well to a comparison. The bacteria in the root canal can be compared to an army entrenched behind high and inaccessible mountains. Through a mountain pass, the apical foramen, this army tries to descend and invade the plain beyond the pass, the periodontal soft tissue and the rest of the body. Another army in the plain guards the pass; it has built trenches and fortifications, the granulation tissue, and tries to prevent the mountain army from progressing farther. The defending army is represented by the white blood cells and other cells of the granulation tissue. Naturally the soldiers of the defending army are grouped closely around the opening of the pass through which the enemy is trying to come; for the same reason the white blood cells accumulate near the opening of the apical foramen.

For a long time there may be no action. Occasionally, a few soldiers of the mountain army, bacteria, descend through the pass, the apical foramen, but they are usually captured and destroyed by the defenders, the white blood cells. Then, suddenly, the mountain army makes a mass attack, and a battle ensues. Such a battle between invading bacteria and body tissues is clinically known as acute inflammation.

The outcome of this battle may vary. The bacteria may win and invade the plain; then the clinical manifestations are an acute dento-alveolar abscess, or even a general septic infection. Or, the defenders, the white blood cells, may be successful; they may overcome the invading bacteria, and afterwards the rest of the mountain army will again be confined to the field above the pass, the root canal, to which the white blood cells have no access.

This parallel can be carried still further. If the attacking mountain army, the bacteria, are eliminated, either by extraction of the tooth or by sterilization of the root canal, the defending army is no longer needed. It is demobilized, the granulations shrink, and the soldiers, the white blood cells, leave and return into the general circulation, perhaps to be used elsewhere to ward off a similar attack. This explains why the apical granulation tissue disappears after the extraction of the infected tooth or after successful root canal therapy.

This comparison, although it has its shortcomings, helps to explain a few observations. It explains why sometimes bacteria are found in granulomas and sometimes not; it illustrates the inaccessible position of the bacteria in an untreated root canal, and also the defense mechanism created because of a flare-up of a chronic peri-

apical inflammation. Likewise, it emphasizes the function of the granuloma, not as something harmful, but as a defense against the spread of infection.

FOCAL INFECTION.

Focal infection of dental origin is usually associated with the problem of chronically infected pulpless teeth; therefore, it will be briefly considered here.

Generally, focal infection is defined as a secondary infection in a distant tissue or organ caused by an invasion of the blood stream by microorganisms from a primary focus. Such a primary focus may be any tissue harboring pathogenic microorganisms. A focal infection results, for instance, if tubercle bacilli are disseminated from a primary focus in a bronchial lymph node to other organs, such as the kidneys. In modern medicine, however, the term focal infection is almost exclusively used for infections produced by streptococci, especially *Streptococcus viridans* and *Streptococcus hemolyticus*.

A focus of infection is a circumscribed and confined area that contains pathogenic microorganisms and that usually causes no clinical manifestations. Most of such foci are located near the body surface or near the orifices of the intestinal, respiratory, or genito-urinary tract. The most common foci harboring streptococci are located in the head; they are infected tonsils, infected teeth, and chronically inflamed nasal sinuses.

Dental infection can be divided into three types, periapical infection (chronic periodontitis), periodontal infection (pyorrhea), and chronic pulpitis. Of the three, the first type, the infected pulpless tooth, is the most important, but the other two must not be overlooked in the clinical examination of patients with suspected focal infection of dental origin (Cook).

The existence of a focus does not *per se* constitute actual focal infection. The bacteria may remain confined within the focus and not spread beyond it. But, potentially, the danger of focal infection exists whenever pathogenic microorganisms have gained a hold in the tissues.

The number of diseases that have been attributed to metastatic spread of streptococci from infectious foci is very large. Rosenow has gone far in citing focal infection as the etiology of a variety of pathological conditions. He particularly emphasized the theory of elective localization, which implies that certain strains of bacteria become adapted to one kind of tissue and retain this specificity. Thus, microorganisms from patients suffering from arthritis, if injected into animals, would cause a preponderance of joint lesions;

microorganisms from ulcerative colitis, lesions in the animal's colon, etc. Rosenow's findings, however, are not accepted by many prominent clinicians and bacteriologists; other research workers have been unable to reproduce many of his experimental results.

It is not the author's intention to enter into a discussion of the vast amount of literature and the extensive controversies concerning focal infection. Only a few of those conditions will be cited here whose relationship to focal infection has been fairly well established.

Perhaps the most important medical problem in connection with focal infection is that of arthritis. Above all, a distinction must be made between the different types of arthritis. In the traumatic and degenerative forms, which are not of infectious nature, no relief or cure can be expected from the removal of possible foci. Only in the non-specific, chronic, infectious type of arthritis is there a possibility of an etiological relationship to infected teeth. Similar restrictions apply to neuritis. Occasionally, patients suffering from neuritis have shown improvement after the elimination of dental infection, but, on the whole, the clinical results are not very encouraging, since neuritis apparently may be due to a number of causes, among which dental infection is a minor one.

What part focal infection plays in the etiology of heart disease is still uncertain. Most internists recommend the elimination of dental infection in cardiac patients, but evidence of actual improvement following such operations is scarce. In children chronic bacterial endocarditis may follow chronic infection of the tonsils or teeth. In diseases of the urinary tract, focal infection is considered an accessory etiological factor that should always be investigated and, if possible, removed.

Chronic diseases of the gastro-intestinal tract, especially chronic ulcerative colitis, are sometimes favorably influenced by the removal of dental foci of infection. Cook reported extensive experimental studies that indicate a definite correlation between streptococci from dental lesions and ulcerations of the colon.

In the eye, inflammation of the iris (iritis) has been found to be due to chronic dental infection in about 12 per cent of all cases. Other inflammations of the eye that are occasionally caused by chronic dental infection are iridocyclitis and uveitis.

Diseases of the skin, especially those grouped under the general term eczema, have occasionally been beneficially influenced by the removal of infected teeth; however, one must be careful not to draw

too far-reaching conclusions concerning the relationship between skin diseases and dental focal infection.

Patients suffering from secondary anemia without any demonstrable cause should be examined for dental infection on the premise that such an infection may be a contributing cause to the anemia. The relationship of dental infection to nervous and mental diseases has no doubt been much overemphasized. With the possible exception of some cases of neuritis, no improvement should be expected from the removal of suspected dental foci in such patients.

The question is often asked: How definite is the correlation between the focus and the secondary infection, and what results can be expected from the removal of a suspected focus? This question is difficult to answer. After a suspected focus has been eliminated one of three things is possible: the patient's symptoms may remain unchanged, or they may abate temporarily, or there may be permanent improvement.

In the first case, if no improvement ensues, the clinical manifestations either were not of focal infection origin, or even if they originally were caused by focal infection, the bacteria in the meantime have become established in their new habitat and are no longer influenced by removal of the primary focus. Temporary improvement is quite common, especially in chronic diseases with frequent remissions, such as arthritis or iritis; it may lead to unwarranted optimism with subsequent disappointment. Occasionally, removal of a focus is followed by permanent improvement, suggesting an actual etiological connection between the focus and the secondary disease. Unfortunately, however, such favorable results happen far less frequently than one might be led to believe from a none too critical survey of the literature on this subject.

The ultimate solution of the focal infection problem lies with the dentist. This solution can be stated with one word: prevention. Some day, perhaps not too far distant, there will be no more infected pulpless teeth, infected pulps, or pyorrhea pockets, not because all teeth thus affected have been extracted, but because dentists and patients will have learned to prevent all these conditions by good care, systematic oral health supervision, early treatment of dental caries and pyorrhea, and operative and prosthetic restorations that do not endanger the vitality of the dental pulp. The progress already made during the past three decades is very encouraging, and at the rate at which the average number of infected pulpless teeth in the mouths of patients is decreasing, focal infection will some day cease to be a serious dental and medical problem.

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CHAPTER IX.

TISSUE CHANGES FOLLOWING ROOT CANAL THERAPY.

ANATOMY OF THE HUMAN ROOT CANALS.

KNOWLEDGE of the anatomy of the human root canals is essential to successful root canal therapy. The investigations of the last two decades have brought forth so much new and basic information on this subject that it seems necessary briefly to describe these recent findings before entering into the discussion of the tissue changes associated with root canal treatment.

The connection between the dental pulp and the circulatory and nervous systems of the jaw passes through the apical foramen. In some teeth this connection consists of a single strand of tissue that passes through one wide, straight apical foramen. Much more frequently, however, the pulp at the root end is divided into several branches that pass through the dentin and cementum as several fine canals.

Variations of the Apical Foramen.—If there is one simple apical opening, a number of variations can be observed, which will be illustrated by means of longitudinal sections through root ends of teeth in the jaw. The simplest form of apex is illustrated in Figure 181. The apical foramen is a continuation of the wide, straight root canal, opening at the very tip of the root. The blood-vessels and nerves of the pulp pass through the foramen and connect through the adjacent bone with the main vessels and nerves of the jaw. The apical foramen itself is formed by cementum; the border between cementum and dentin, the dentino-cemental junction, has no significance whatsoever in the form or diameter of the apical foramen, nor is it necessarily true that the foramen is the narrowest point in the entire root canal. Sometimes the root canal becomes narrower in its apical part; frequently it has a uniform width throughout, and occasionally it even becomes wider at the apical foramen. This is important, since there seems to be a rather widespread opinion among practitioners that the foramen is always the narrowest point in the course of the root canal and that thus it may be recognized clinically by probing the canal.

The simple, straight form of apical opening illustrated in Figure 181 is not very common. In histological sections of various human

teeth of different ages this type of foramen was found in only 22 per cent, or about one-fifth of several hundred teeth. More frequently the apical part of the root canal has a certain degree of curvature; either the entire root end is curved, or the root end is straight and the canal is curved, so that the foramen is not located at the extreme end of the root. This is illustrated in Figures 182 and 183. In Figure 182 the foramen is found at the very tip of the root, but the entire

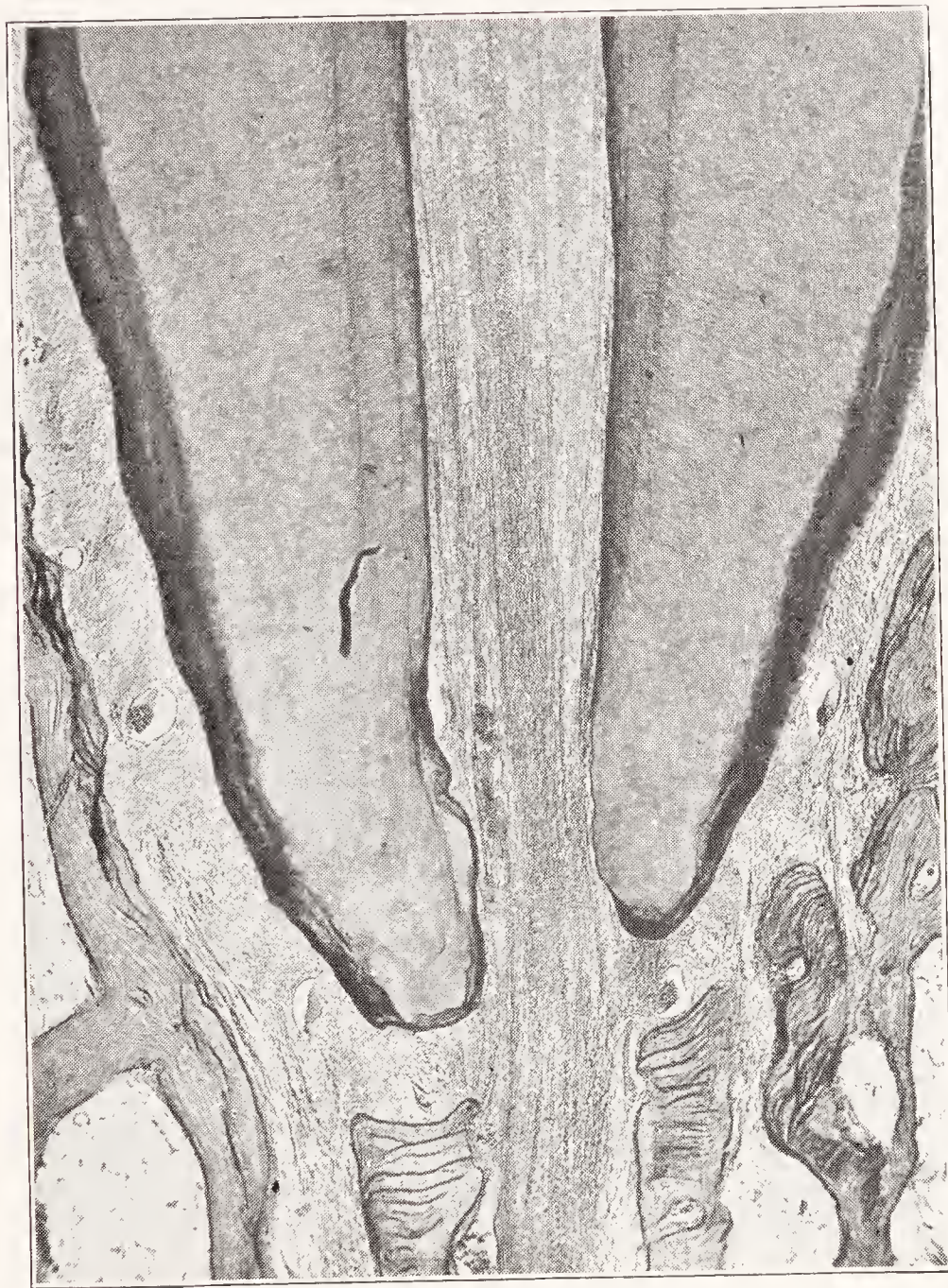


FIG. 181.—Apex of upper central incisor. Straight, wide root canal. Slight constriction of the canal at the apical foramen. (Coolidge, Jour. Am. Dent. Assn.)

apical portion of the root is curved. In Figure 183 the root end is straight, but because of a deviation of the root canal the apical foramen is located on the side of the root tip.

Ramifications of the Root Canal.—In the majority of human teeth the root canal does not run as one single canal through the dentin, but separates into two or more canals near the root end. These

divisions of the root canal are called apical ramifications. Other branches run at right angles from the main canal to the root surface; they are called lateral branches. The frequent occurrence and clinical importance of these variations in structure have not been recognized until recently. Hess and his coworkers have made the most complete study up to this time of the anatomical variations in human root canals in different groups of teeth.



FIG. 182.—Apex of mesial root of lower second molar. The root end is slightly curved toward the distal side. (Coolidge, Jour. Am. Dent. Assn.)

The following technique was used by Hess for his original investigation: A large number of intact human teeth was collected and recorded separately as to kind of tooth and age of patient. The pulp chambers were opened and the organic content of the pulp canals removed by maceration and rinsing. After the canals had been cleaned, unvulcanized soft rubber was forced into them from the pulp chamber under pressure until the rubber completely filled all spaces within the teeth. Then the teeth were vulcanized. By sub-

jecting them to the action of a strong acid, the entire tooth structure was dissolved. The remaining vulcanite casts were exact reproductions of all hollow spaces inside the teeth. These vulcanite casts were studied, and the number and distribution of the ramifications recorded. A few of the specimens of Hess will be reproduced



FIG. 183.—Apex of a cuspid. The root end is straight but the root canal is curved. As a result, the apical foramen is not located at the root end but on the side of the root. (Coolidge, Jour. Am. Dent. Assn.)

here, as they give a clear picture of the inside anatomy of human teeth.

Figure 184 shows the vulcanite casts of four upper central incisors. Two of the illustrated teeth have simple root canals without branches; of the other two, one has several small apical branches, and the other a canal at right angles to the main canal. Similar conditions are found in upper lateral incisors (Fig. 185).

In the lower incisors and cuspids, a common anatomical variation

is the occurrence of two main canals that may separate either at the level of the pulp chamber or further apically. Hess found such divisions in 37 per cent of lower incisors, and in 43 per cent of lower cuspids (Figs. 186 and 187).

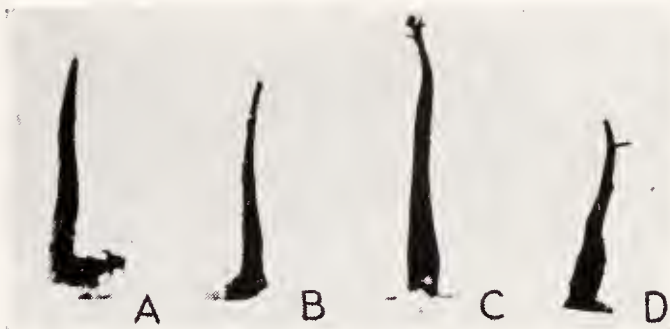


FIG. 184



FIG. 185

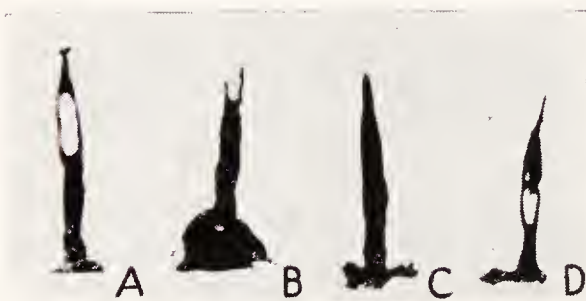


FIG. 186



FIG. 187

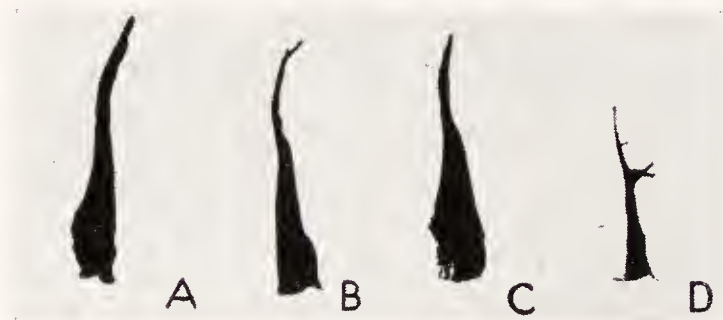


FIG. 188

FIGS. 184-193.—Anatomy of the root canals of human permanent teeth. (Hess, Schweiz. Vrtljschr. f. Zhk.)

FIG. 184.—Vulcanite casts of the root canals of the upper central incisors. *A, B*, single straight canal; *C*, apical ramifications; *D*, lateral branch.

FIG. 185.—Upper lateral incisors. *A*, single straight canal; *B, C, D*, lateral branches.

FIG. 186.—Lower central and lateral incisors. *A, B, D*, partial division of the main canal; *C*, single wide canal.

FIG. 187.—Lower cuspid. *A, B*, single wide canal; *C, D*, two canals.

FIG. 188.—Upper cuspid. *A*, single wide canal; *B*, apical ramification; *C, D*, lateral branches.

The upper cuspid has a straight root canal that is much wider than that of the upper incisors. There are apical ramifications in about 25 per cent of upper cuspids; sometimes a lateral branch extends at a right angle from the main canal in its apical portion (Fig. 188).

In the upper bicuspid the anatomy of the root canals is rather complicated. According to the investigations of Hess, only 19.5 per cent, or one-fifth, of all upper first bicuspid have one single,

wide canal (Fig. 189, *A*). The remaining four-fifths have two canals with numerous fine branches, some of which connect the two canals with each other (Fig. 189, *E*), while others form apical ramifications (Fig. 189, *C*, *D*). In the upper second bicuspid the division into two canals is less frequent, 56 per cent, or more than one-half, having one canal (Fig. 190). Apical ramifications are very common.

Lower bicuspids (Fig. 191) usually have one root canal, a division into two canals being more frequent in the second bicuspid than in the first.



FIG. 189

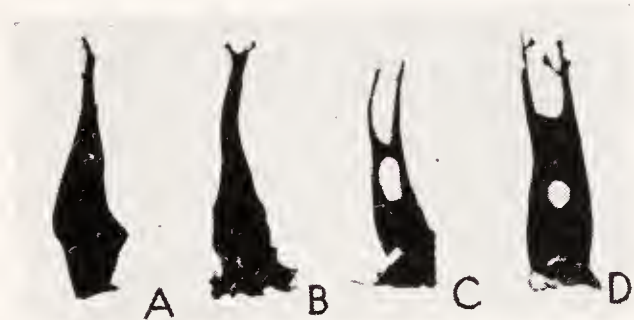


FIG. 190

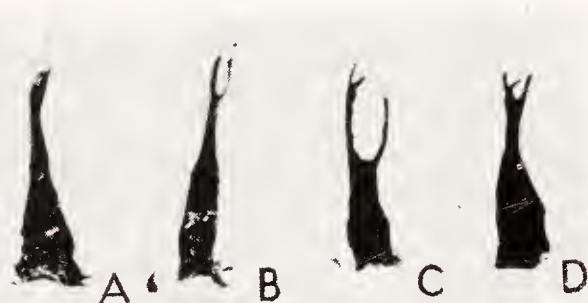


FIG. 191

FIG. 189.—Upper first bicuspid. *A*, single wide canal; *B*, *C*, *D*, different forms of two canals; *E*, connections between the two canals.

FIG. 190.—Upper second bicuspid. *A*, single wide canal; *B*, single canal with apical ramifications; *C*, two canals; *D*, partial division of the canal with apical ramifications.

FIG. 191.—Lower first and second bicuspids. *A*, *B*, *D*, apical ramifications; *C*, partial division of canal.

Lower molars vary in the number of root canals (Hess):

	1 canal.	2 canals.	3 canals.	4 canals.
Lower molars	0.3 per cent	17.7 per cent	78 per cent	4 per cent

Apical ramifications are frequently found in all roots (Fig. 192). Mesial root canals are usually connected to each other by fine branches running horizontally from one main canal to the other (Fig. 192, *E*). The lower third molar usually has two canals with comparatively few ramifications.

The upper first and second molars usually have three canals; although a division of the mesio-buccal canal sometimes makes four canals (Fig. 193, *D*, *F*). Apical ramifications were found in more

than one-half of all upper molars examined. The root canals of the upper third molar vary more than those of any other tooth; anywhere from one to five can be found.

In deciduous teeth the anatomical conditions are similar. Hess showed that deciduous incisors and cuspids, as a rule, have short, wide, single root canals with occasional ramifications. Deciduous molars, however, have complicated root canals; both upper and lower molars frequently have four canals and, in addition, many ramifications.

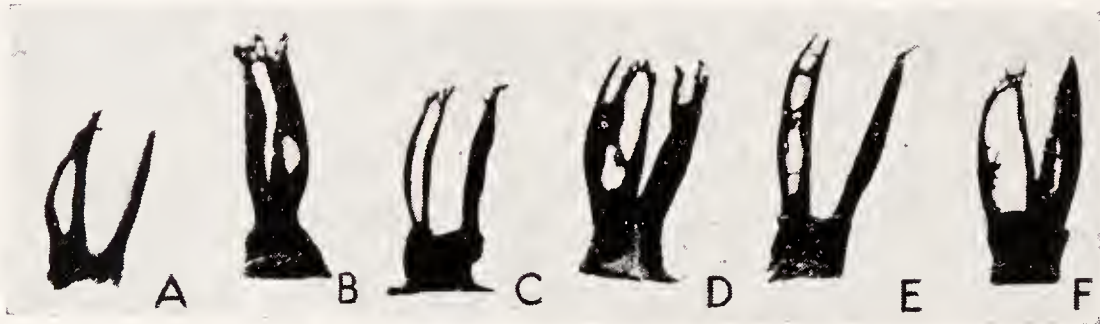


FIG. 192



FIG. 193

FIG. 192.—Lower first and second molars. *A, C*, three canals with apical ramifications; *E*, connections between the two mesial canals; *B, F*, wide canals with apical ramifications; *D*, four canals.

FIG. 193.—Upper first and second molars. *A, B*, three canals with apical ramifications; *C*, two canals; *D, E*, irregularities of the buccal canals; *F*, four canals.

RELATIVE FREQUENCY OF APICAL RAMIFICATIONS AND LATERAL BRANCHES IN HUMAN PERMANENT TEETH. (AFTER HESS.)

Kind of tooth.	Apical ramifications, per cent.	Lateral branches, per cent.
Upper central incisor	25	21
Upper lateral incisor	31	22
Upper cuspid	25.5	18
Lower central and lateral incisor	21.6	10
Lower cuspid	39	12
Lower first bicuspid	44	17.3
Lower second bicuspid	49	20
Upper first bicuspid	41	18
Upper second bicuspid	50	19
Upper first and second molar	67	16
Upper third molar	80	23
Lower first and second molar	73	13.5
Lower third molar	10	6

The average frequency of irregularities in the structure of the root canals of human permanent teeth was compiled by Hess in a table that is reproduced here in abbreviated form. He differentiated between apical ramifications (terminal branches of the main canal) and marrow canals (lateral connections between the main canal and the periodontal membrane elsewhere in the root). In this table apical ramifications and lateral branches are recorded separately. Several thousand teeth of patients of all ages were used for these investigations; the figures present the averages for different ages. In very

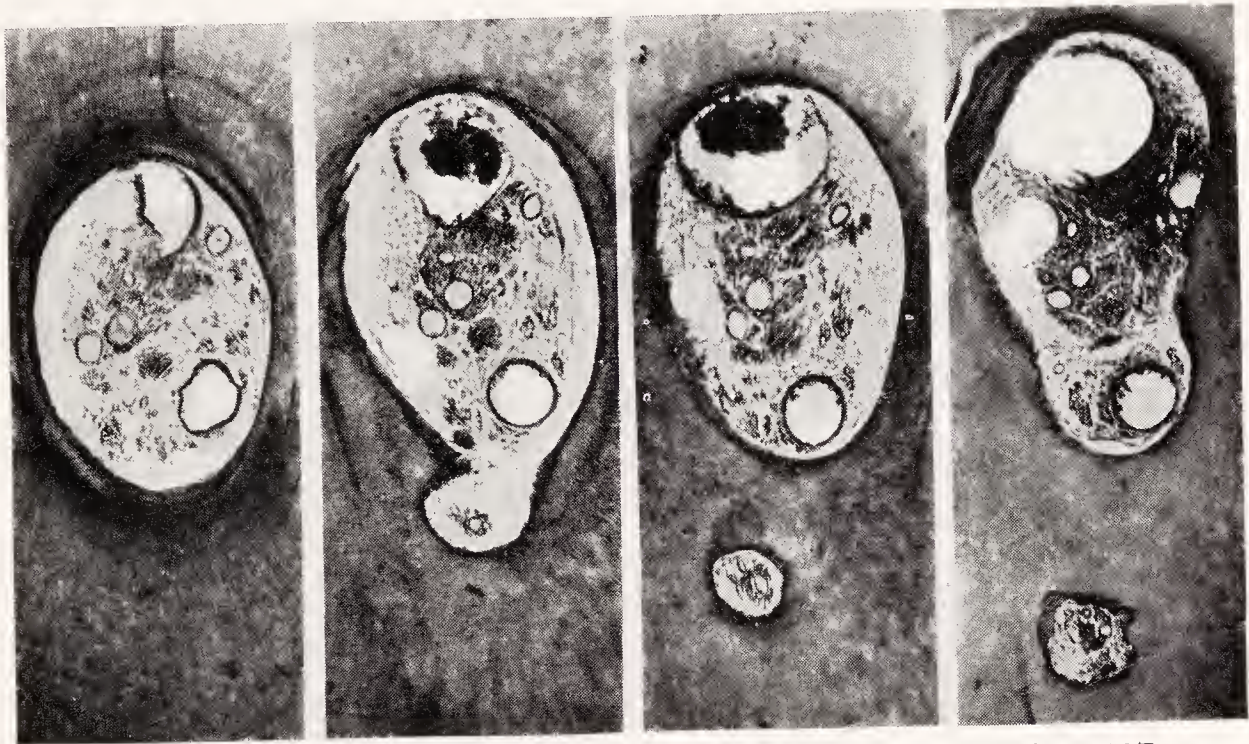


FIG. 194

FIG. 195

FIG. 196

FIG. 197

FIGS. 194-197.—Successive cross-sections through the apical portion of the root of a lower first bicuspid.

FIG. 194.—Single round canal.

FIG. 195.—Branching of apical ramification.

FIG. 196.—Main canal and apical ramification separated.

FIG. 197.—Branching of another apical ramification from the main canal.

young teeth the number of branches and ramifications is small. Between the ages of twenty and forty years the number is largest. Later in life some are obliterated by progressing dentin formation.

In histological specimens these ramifications can easily be studied in serial sections. Figures 194 to 197 illustrate cross-sections through the apex of a human lower first bicuspid at different levels. In Figure 194, a cross-section through the root in its lower third, the root canal is single; slightly nearer the apex (Fig. 195) the canal changes its shape; here a smaller branch can be seen leaving the main canal. In Figure 196 the lateral branch has become completely separated from the main canal. The latter splits again as it approaches the root tip (Fig. 197), showing that there are three apical foramina in this apex.

In longitudinal sections through human root ends, lateral canals are found to branch from the main canal at different levels. Apical ramifications of the pulp canal near the foramen in a lower bicuspid are illustrated in Figure 198. In this tooth the root canal branches into three strands.



FIG. 198.—Root end of lower second bicuspid of an adult. *MC*, main canal; *AR*, apical ramifications; *PM*, periodontal membrane; *AB*, alveolar bone; *FM*, fat marrow of alveolar process.

TISSUE CHANGES FOLLOWING REMOVAL OF THE PULP AND TREATMENT OF THE ROOT CANAL.

The tissue changes that take place if a vital pulp is removed from a root canal will now be considered. The knowledge of these changes has been gained partly from human specimens and partly from animal experiments. Pulp removal following exposure of the pulp by caries will be considered first. The dentist, after removing the soft, decayed dentin, finds the pulp exposed; he administers a local anesthetic, applies the rubber dam, and removes the pulp tissue from the root canal by means of a barbed broach. By this operation the pulp strand is torn off near the apical foramen, creating a wound on the surface of the remaining tissue. The location of this wound depends upon local circumstances. In a root like the one illustrated in Figure 181, the tissues most probably will be severed at the foramen or beyond it, in the apical periodontal tissue. If the root is like those illustrated in Figures 182 and 183, the pulp wound will usually be located at the point where the canal curves. The removal of the

pulp invariably is followed by a hemorrhage from the torn pulp vessels. After the hemorrhage has subsided, a clot of fibrin forms on the surface of the pulp wound.

The prognosis of this wound depends principally upon two factors, namely, the presence or absence of infection and the condition of the soft tissue in the region of the pulp wound.

Infection.—Every wound is a good medium for the invasion and development of pathogenic microorganisms. This is especially true of a lacerated wound of the type that results from pulp removal, with its torn surface and considerable tissue injury. Therefore, if pathogenic microorganisms gain access to the pulp wound, the chances are that they will spread rapidly, multiply, and cause an infection of the pulp wound and the periodontal tissue. Maintenance of strict asepsis during all manipulation in the root canal is essential to prevent infection of the pulp stump. The result of the root canal operation will depend largely upon the care exercised in this respect.

Condition of the Remaining Tissue.—The healing processes after pulp removal occur in the tissue adjacent to the point where the pulp was severed. The vitality of these tissues must be maintained in order to make healing possible. Great harm can be done to the pulp wound by the use of too powerful drugs. In the early days of root canal therapy concentrated acids were among the drugs recommended and used. Since then the importance of the apical tissues in the successful outcome of any root canal operation has become known, and all of these tissue-destroying drugs have been ruled out. The enlargement of the canals, formerly done by acids, is now performed with sterile, clean-cutting instruments, and the necessity of using strong disinfectants has been overcome by employing careful asepsis. Thus, the living tissues in the depths of the root canals are kept alive and can fulfill their reparative functions.

Attention should be called to the parallel development in surgery and root canal operations. When knowledge of bacteria was first introduced into the practice of surgery, strong antiseptic solutions, such as phenol, were generally used on instruments, tissues, and dressings. Since then the injurious influence of such drugs upon the living cells has become known; asepsis has taken the place of antiseptics, and instead of fighting bacteria in the field of operation, they are kept out by aseptic measures. No modern surgeon would apply a powerful drug to living tissue that he expects to heal.

After pulp removal, a sterile, non-irritating dressing is sealed into the root canal. The pulp wound continues to discharge a serous exudate for a day or two; at the same time, an accumulation of white

blood cells is found near the wound surface, a reaction that occurs in the neighborhood of every wound during the early stages of repair. The white blood cells form a protective wall between the wound and the rest of the body; by phagocytosis they dispose of the cell débris present wherever injury to the tissue has occurred and help to destroy any bacteria that may have reached the wound.

The exudation and round-cell infiltration following removal of the pulp are responsible for the slight soreness frequently encountered following this operation. As healing progresses and exudate and infiltrations disappear, this soreness subsides.

Soon the discharge from the wound stops; a clot of fibrin forms, and under this protective cover fibroblasts begin to proliferate and initiate scar formation. Finally a connective-tissue scar is formed at the point where the pulp was severed.

Similar changes take place if the pulp is removed following devitalization with arsenic trioxide. The comparative merits and demerits of the two methods of pulp removal—under infiltration anesthesia and following the use of arsenic—are still being debated; both methods apparently give equally good results in the hands of skillful and conscientious operators.

Quite different changes take place, however, if pathogenic microorganisms have gained access to the pulp wound, or if a strong, harmful drug has been used in the root canal. The round-cell infiltration increases rapidly, indicating the reaction of the tissue to advancing infection. Instead of the formation of a scar on the surface of the pulp stump, more and more tissue breaks down; soon the periodontal membrane around the foramen is involved, and periodontitis develops—in clinical terms, the root canal operation has been a failure.

The portion of the root canal from which the pulp tissue has been removed is filled with a root filling material, usually guttapercha. The type of material used does not seem to be of great importance so long as the foreign substance is well tolerated by the tissues and has physical qualities that make its manipulation and practical application possible. Good tissue tolerance has been shown for guttapercha as well as for silver or ivory. Because of advantages in manipulation, guttapercha is most commonly used at the present time.

After removal of a pulp and filling of the root canal, the formation of a connective tissue scar at the root end is almost invariably followed by a deposition of cementum in this area. From findings in human teeth that were extracted a few months after the root canals were filled, it appears that the deposition of cementum begins several

weeks after the operation; from this time on, the amount of cementum increases slowly but steadily. Cementum has a tendency to obliterate all spaces that are left after a root canal is filled. That such spaces may exist is obvious from the study of specimens like the ones shown in Figures 184 to 193. Only in a root canal of the type shown in Figure 181 is a complete root filling possible; even when the filling extends to the apical foramen according to the radiograph, the root canal is usually either slightly overfilled or underfilled. Usually living pulp tissue is left in parts of the main canal or ramifications that are inaccessible to root canal instruments. After the transformation of the pulp tissue into connective tissue, a reparative formation of cementum takes place; finally the cementum obliterates the lumen of the canal, thus forming a far better root canal filling than any foreign material.

The deposition of cementum following root canal therapy was first observed and described in this country by Davis and Grove. In Europe, Swiss investigators led in this particular field of research (Gysi, Hess, O. Mueller). Recent American publications by Blaney, Coolidge, Hatton, Moen, Skillen, and others have dealt with the same subject. All reported that cementum is deposited upon the root end and in the unfilled part of the canal, with the tendency to cover the root filling material and to obliterate any lumen still present in the apex. They also showed that this process occurs only in the absence of infection.

A few specimens will be described that were obtained from human teeth whose pulps had been removed. In order to interpret the tissue changes found at the apices of these teeth, these changes can be compared with the changes following bone fracture, a point brought out by Hatton. He said: "In the healing of the broken bone there are found the same stages that are found in the apical region of treated and filled teeth: (1) A stage of destruction or dissolution of the products of the injury, such as blood, damaged soft tissues, and a certain amount of bone; (2) a stage of repair, characterized by the formation, first of a fibrous callus, and later by the production of new bone or the bony callus; and (3) the resting stage." In pulpless teeth the first stage is represented by the reaction of the periodontal tissues to the trauma of pulp removal; during this stage resorptions may occur on both root surface and alveolar bone, hence, the frequent observation of repaired resorptions at the apices of healed pulpless teeth. After the results of the initial trauma have been overcome, scar formation takes place, beginning with the formation of fibrous connective tissue, which later is replaced by

bone and cementum. This process of hard tissue formation at the apex is continued until the root end has reached what Hatton terms the "resting stage" during which the pulpless tooth, without significant tissue changes, continues to function like a bone with a well-healed fracture.

FINDINGS IN HUMAN TEETH ON WHICH ROOT CANAL OPERATIONS HAD BEEN PERFORMED.

The most valuable source of knowledge of tissue reactions following root canal operations is extracted human teeth with known clinical histories. There are, of course, other approaches to this problem, the most important of which is experimental root canal therapy in animals, but because of the possible differences in tissue reaction between man and animals, the results obtained in animals are of limited value. Moreover, all animal experiments are of relatively short duration, while it is possible to examine human teeth that were treated fifteen or twenty years before extraction. Since the root end of a pulpless tooth heals slowly, the time factor is of great importance. Animal experimentation has the advantage that teeth and jaw together can be examined in their original relationship, while in extracted human teeth this relationship is lost. However, it has been found that the great majority of extracted human teeth retain enough periodontal tissue on the root surface and at the foramen to allow drawing definite conclusions as to the condition of the root end. The following illustrations are taken from such specimens, and in every one of them the changes that resulted from the loss of the pulp can be clearly recognized.

Tissue Changes in the Main Canal Following Root Canal Filling.— Only in a small percentage of teeth with root canal fillings that radiographically seem entirely to fill the canal does the filling material end level with the apex. Most roots are slightly underfilled, the length of the unfilled portion of the main canal varying between 0.5 and 2 mm., and containing fibrous connective tissue, which may be either a remnant of the original pulp tissue or periodontal connective tissue that proliferated into the open apical portion of the root canal. The connective tissue has a tendency to form cementum, which is deposited in layers on the wall of the pulp canal. Figure 199 illustrates an early stage of cementum deposition. The guttapercha ends about 1.5 mm. short of the apical foramen; the remaining portion of the root canal is filled with fibrous tissue. The cementum covering the outer surface of the root end continues through the

foramen into the canal; its thickness decreases toward the root canal filling. Six months had elapsed between the removal of the pulp and the extraction of this tooth.

In Figure 200, a lower bicuspid, a similar condition is illustrated. Here more cementum has been deposited because more time had elapsed since the root canal operation was performed. The gutta-percha cone ends about 1 mm. above the apex. The apical portion of the root canal contains well-vascularized connective tissue that

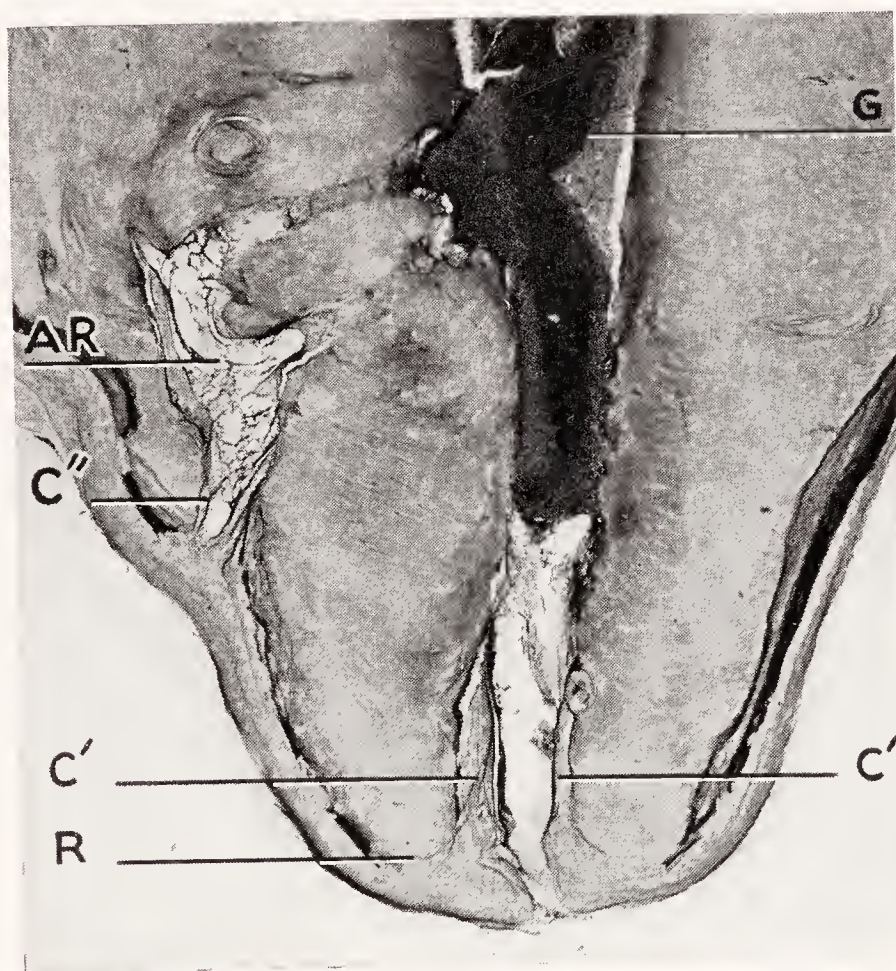


FIG. 199.—Condition of root end six months after pulp removal and root canal filling. Buccal root of upper molar. *G*, gutta-percha filling in main canal; *AR*, apical ramifications of the main canal; *R*, areas of resorption in cementum and dentin covered by new layers of cementum; *C'*, cementum extending upward into the unfilled portion of the main canal; *C''*, cementum extending into the apical ramifications. (Coolidge, Jour. Am. Dent. Assn.)

is entirely free from inflammation; cementum has been deposited over all of the wall of the root canal. The surface of the gutta-percha cone is covered by a layer of uncalcified cementum. The right wall of the root canal runs in a straight, smooth line that can be explained only by the action of a cutting instrument, a broach or reamer. During enlargement of the root canal the operator apparently reached the apical foramen; the root canal was filled, but the filling material did not reach the foramen, and the apical portion of the canal was subsequently covered with cementum.

The condition of a root end eight years after the root canal

operation is illustrated in Figure 201. The obliteration of the unfilled apical part of the canal is almost complete; the connective tissue in the canal has been reduced to a thin strand, and several layers of cementum cover the dentinal walls. The surface of the dentin forming the wall of the root canal was resorbed before cementum deposition took place; evidently resorption preceded tissue repair in the canal. A small piece of guttapercha, which became dislodged close to the apical foramen, has been completely embedded in the

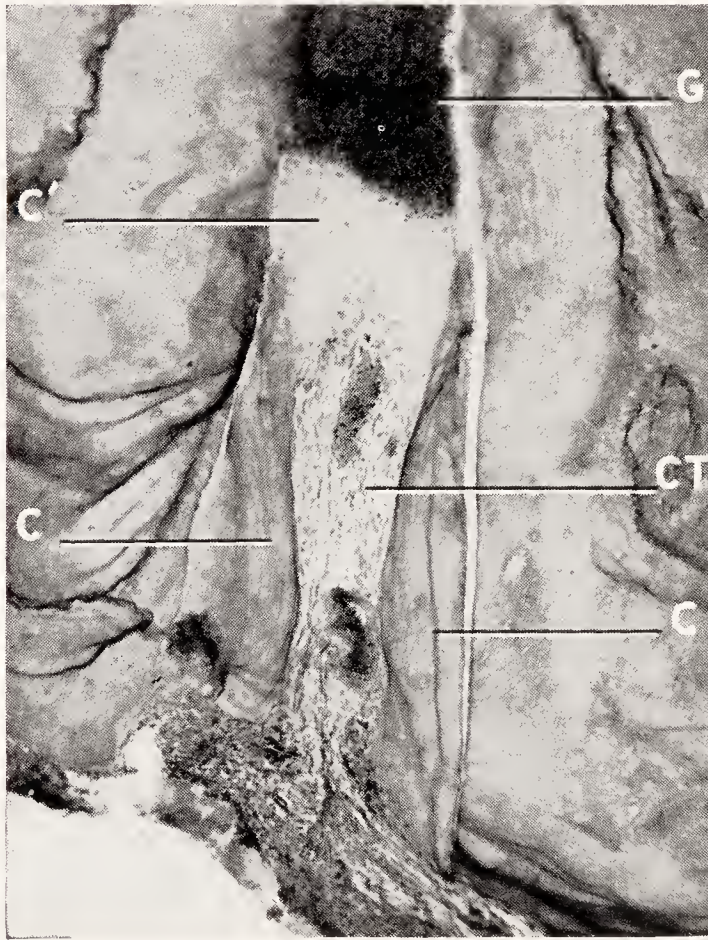


FIG. 200.—Cementum deposition in the apical portion of the root canal several years after pulp removal and root canal filling. *G*, guttapercha; *C*, cementum covering the wall of the apical portion of the root canal; *C'* cementoid (newly formed uncalcified cementum) deposited upon the guttapercha; *CT*, connective tissue in the root canal.

layers of cementum that surround the remaining thin strand of connective tissue.

If the guttapercha reaches the apical foramen and ends level with the root surface, a fibrous capsule is formed, bridging the foreign material; later on, new cementum is deposited on the root surface and sometimes also directly upon the guttapercha surface (Fig. 202). The latter observation is very important. It shows that guttapercha as a root filling material is well tolerated by human connective tissue; not only will tissue grow against the guttapercha without any evidence of irritation, but also cementum is deposited directly upon the guttapercha surface. Such findings indicate that theoretical

objections against guttapercha as a foreign body are not substantiated by the actual reaction of living human tissue to this material.

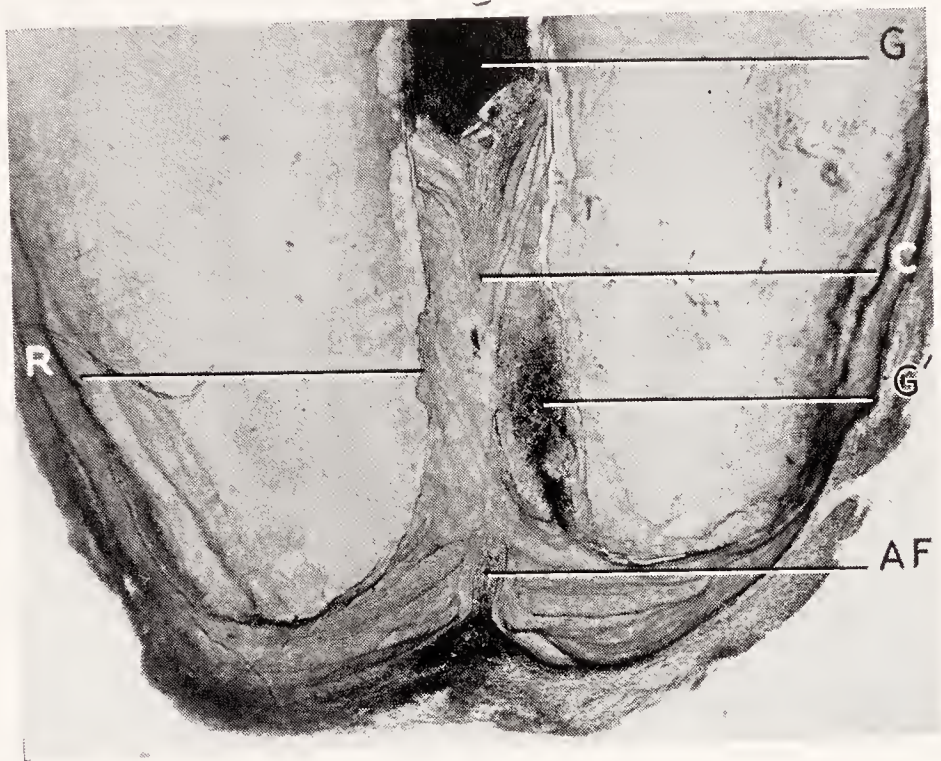


FIG. 201.—Advanced stage of cementum deposition in the apical portion of the root canal. The pulp was removed and the canal filled eight years before the extraction of the tooth. *G*, guttapercha; *C*, cementum in the apical portion of the root canal; *R*, resorption in the wall of the root canals filled by cementum; *G'*, small particle of guttapercha completely embedded in cementum; *AF*, apical foramen. (Coolidge, Jour. Am. Dent. Assn.)



FIG. 202.—Deposition of cementum upon root end and root canal filling. Lingual root, upper molar; treated and filled seven years before extraction. *G*, guttapercha; *C*, original cementum on root surface; *NC*, newly deposited cementum covering root surface and end of guttapercha cone; *PM*, periodontal membrane.

In an examination of the periodontal tissue covering the root end of the specimen, only fibroblasts and connective tissue fibrillæ are found; no evidence of inflammatory reaction is present.

If the root filling material protrudes slightly beyond the apical foramen, it is covered with a dense, fibrous, connective tissue capsule (Fig. 203), which is attached all around the apical foramen to a newly

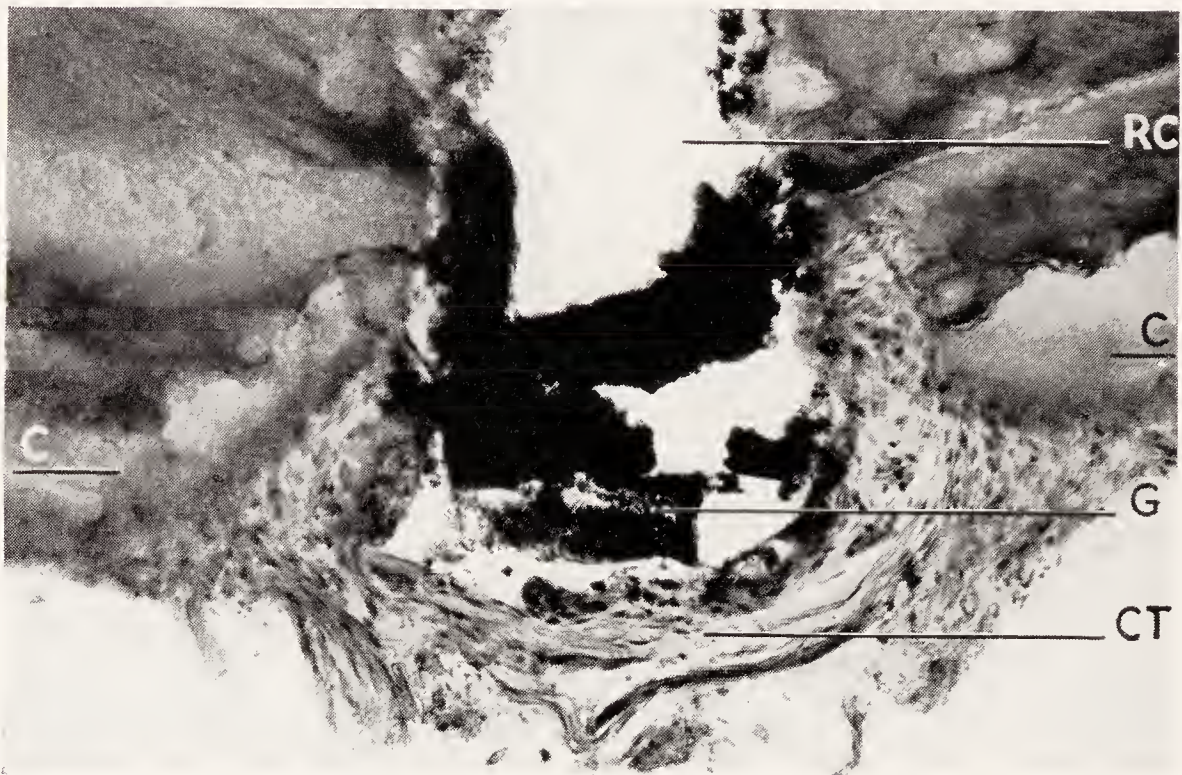


FIG. 203.—Root end with slightly overfilled root canal. Lower bicuspid. *RC*, root canal; *G*, guttapercha; *CT*, connective tissue capsule surrounding the end of the root canal filling; *C*, deposits of new cementum on either side of the root canal.

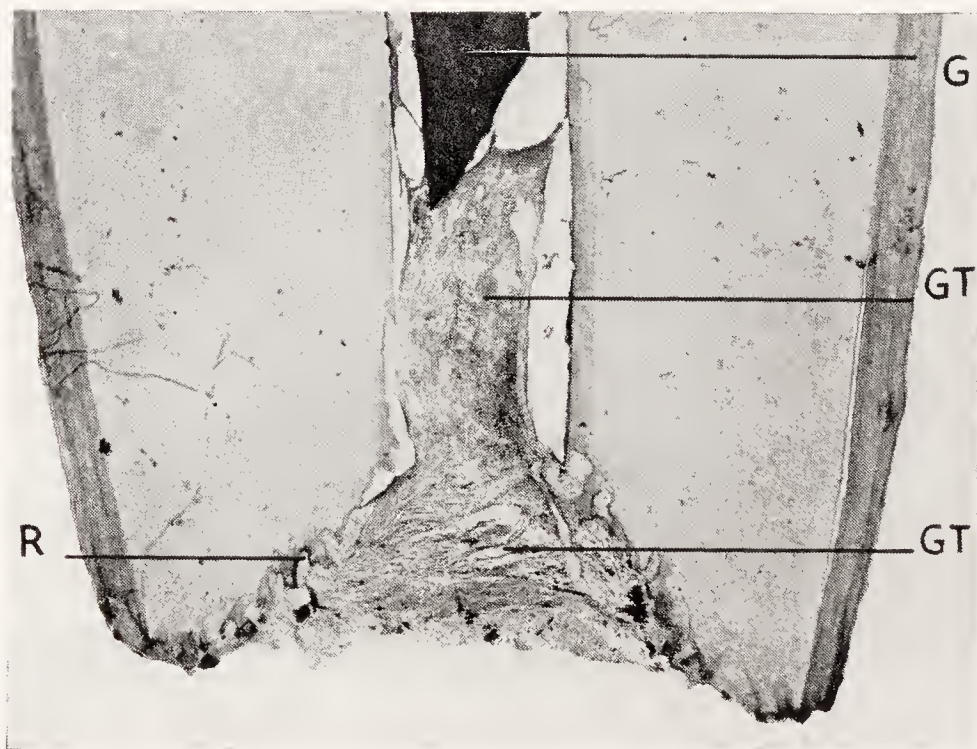


FIG. 204.—Infected root end. Upper central incisor. *G*, guttapercha; *GT*, granulation tissue in the root canal and at the apical foramen; *R*, resorption of root end. Notice the absence of cementum formation in the root canal and at the apical foramen.

deposited layer of cementum. In the tissue of the capsule, giant cells are sometimes found that are comparable to the foreign-body giant cells in the neighborhood of silk sutures or other foreign mater-

ials implanted or inserted into the tissues. No inflammatory cells can be found in the periapical connective tissue of non-infected teeth.

An entirely different microscopic picture is encountered in root ends of infected pulpless teeth. Here the typical deposition of cementum inside or across the apical foramen is never found; instead, granulation tissue develops at the root end. The lumen of the root canal is not reduced; on the contrary, the canal may be widened by resorption without evidence of reparative changes. Such a condition is illustrated in Figure 204, which was taken of the root end of an upper incisor that, radiographically, showed resorption of root end and surrounding bone. The guttapercha root canal filling ends about 2 mm. above the apical foramen; the latter is funnel-shaped as a result of resorption of the surrounding dentin. The unfilled portion of the root canal is filled with granulation tissue with a large number of inflammatory exudate cells (polyblasts). The granulation tissue has grown against the walls of the canal and against the guttapercha cone without any deposition of cementum. A comparison of Figure 204 with Figure 201 reveals the basic difference between tissue changes in the presence and in the absence of infection.

Another case of infection in a tooth with a root canal filling is illustrated in Figure 205, showing the distal root of a lower molar. The apical foramen has been widened by resorption; densely infiltrated tissue fills the space between the wall of the foramen and the guttapercha root canal filling. Farther distant from the infected root canal, cementum has been deposited upon the root surface; from this new layer of cementum, strands of connective tissue extend across the root opening, forming a capsule over the granulation tissue. Cementum formation is completely absent in the inflamed area inside of the apical foramen. In a higher magnification of the tissue in the root canal, the cell types that are characteristic of chronic inflammation can be recognized: plasma cells, lymphocytes, and large phagocytic cells (polyblasts).

Tissue Changes in the Lateral Branches and Apical Ramifications Following Root Canal Filling.—Ever since the existence of the various branches and ramifications of the human root canals became known, these structures have been considered of great significance in the problem of root canal therapy. It is evident that no technique can insure a mechanically perfect root filling in a canal of the form illustrated, for example, in Figure 198. Such irregularities and ramifications of the root canal cannot be filled completely. Fortunately, however, this is by no means necessary. Microscopic

findings in extracted teeth with clinically well-filled, non-infected main canals prove that the tissues take care of the remaining unfilled lateral branches and apical ramifications. All of these fine canals



FIG. 205.—Infected root end. Mesial root, lower molar. *G*, guttapercha; *GT*, granulation tissue in root canal; *AF*, apical foramen widened by resorption of the walls; *C*, deposits of cementum in the periphery of the apical foramen; *CT*, connective tissue capsule extending across the apical foramen. Notice the absence of cementum at the apical foramen and in the root canal.



FIG. 206.—Opening of a lateral branch of the same root the apex of which was illustrated in Figure 202. Ingrowth of cementum from the root surface into the lateral canal. *D*, dentin; *C*, cementum of root surface; *PM*, fibers of periodontal membrane attached to the root surface; *C'*, cementum deposition in the lateral canal; *CT*, strand of connective tissue and capillaries entering the lateral canal.

contain living tissue that remains vital and forms cementum, which eventually may completely obliterate these lateral canals.

An early stage of reparative cementum formation in the apical

ramifications of the main canal is illustrated in Figure 199. The fine branches contain connective tissue; an ingrowth of the cementum on the root surface into the opening of the ramifications has taken place, reducing their width. A more advanced stage of cementum deposition was observed in a fine lateral branch of the tooth the apex of which is shown in Figure 202. The continuation of the cementum into the lateral canal is plainly visible in Figure 206; the tissue in this canal as well as the surrounding periodontal connective tissue is free of any inflammatory reaction.

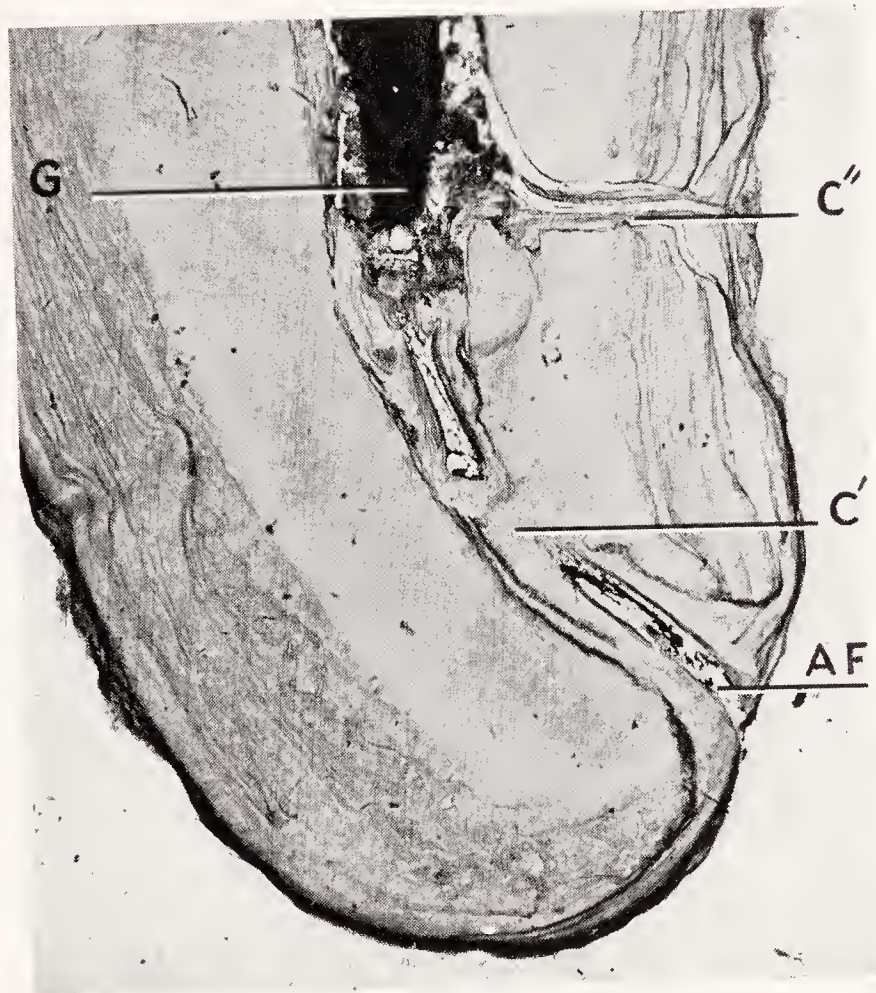


FIG. 207.—Root end of lower molar. Curvature of main canal, lateral branch *G*, end of guttapercha root filling; *C'*, cementum deposition in the curved apical portion of the main canal; *C''*, cementum deposition in the lateral branch; *AF*, apical foramen of the main canal, greatly reduced in width by newly formed cementum. (Coolidge, Jour. Am. Dent. Assn.)

A root end in which reparative cementum deposition has occurred in the curved apical portion of the main canal and in a lateral branch is illustrated in Figure 207. The guttapercha cone ends approximately at the level of the lateral branch; from here to the apex both the main canal and the lateral branch are filled with cementum, leaving only a thin strand of living fibrous tissue in the center of each canal. Eventually the lateral canals may become completely obstructed by cementum (Fig. 208); then the cementum on the root surface is continuous over the former openings of the ramifications.

The same changes that occur in inaccessible lateral branches can be expected at the apical end of main canals that are inaccessible to root canal instrumentation because of denticles or curvature of the root end. The remaining tissue will form cementum, and eventually lead to the obliteration of the apical portion of the canal, provided infection has been kept out.

Changes in the Apical Tissues Following the Treatment of Infected Pulpless Teeth.—In Chapter VIII the histopathology of the apical tissues of teeth with decomposed pulps and infected root



FIG. 208.—Complete obliteration of the opening of a lateral branch. *G*, gutta-percha in the main canal, some of which has entered the lateral branch; *CT*, connective tissue remnants in the lateral canal; *C*, cementum on the root surface covering the opening of the lateral canal in a continuous layer.

canals was described. A radiograph almost always reveals an area of bone destruction around the apex of such teeth. The treatment is a rather difficult problem. The dentist faces the question: Should an attempt be made to save such teeth? Microscopic findings justify such an attempt. Not only do large areas of periapical bone destruction disappear after proper root canal treatment, but also the same reparative and healing processes can occur in formerly infected teeth as in non-infected teeth after the removal of the pulp. However, the treatment of a tooth with a decomposed or infected pulp is not always successful; there is a decided difference between the condition present in a tooth after removal of a vital pulp and that in a tooth with a decomposed pulp and periapical infection. If a living pulp is removed, the chances for undisturbed healing are the same as those in any other properly performed dental operation. If the tooth has a decomposed pulp and infected periapical tissues, the prognosis is uncertain.

The difficulty in treating teeth with decomposed pulps seems to be failure to gain access to all parts of the root canal and to the diseased area beyond the apex. In discussing the tissue changes following removal of a vital pulp, it has been shown that those parts of the pulp canal that are inaccessible to instrumentation are eventually obliterated by formation of cementum (Figs. 206 to 208). In pulpless, infected teeth, such inaccessible parts (ramifications, curved canals) continue to act as a source of infection. If a tooth with a configuration of the apex of the type illustrated in Figure 182 or Figure 183 were infected, the curved root end would make



FIG. 209.—Root end of lower cuspid. Chronic periapical infection treated and root canal filled nine years before extraction. *G*, guttapercha in root canal; *G'*, particles of guttapercha dislodged beyond the apical foramen; *PM*, periodontal membrane. (Coolidge, Jour. Am. Dent. Assn.)

access to the periapical region difficult, perhaps impossible; if the root canal had lateral branches, cleaning and filling the main canal would not affect the bacteria in these ramifications. Yet it is a common clinical experience that large areas of bone destruction around the apices of pulpless infected teeth do disappear completely after successful root canal treatment, and that afterwards the periodontal space appears normal radiographically. Such teeth, when examined under the microscope, show no inflammation of the periodontal tissues; cementum has been deposited on the root end, and the connective tissue surrounding the root is free of inflammation. Such a root is illustrated in Figures 209 and 210. Nine years before extraction, this tooth, a lower cuspid, showed an imperfect root canal filling and an area of bone destruction around the apex. At that time the root canal was opened, cleaned, and filled; sub-

sequently the bone around the apex regenerated. The tooth was observed for nine years and then extracted to make room for an artificial restoration. The microscopic examination showed a root filling extending all the way to the apex. A layer of connective tissue remained attached to the root end after extraction; it was free of any round-cell infiltration and was closely adapted to the root filling material (guttapercha). The dentin surface at the root end shows evidence of previous resorptions that were repaired by deposition of cementum.

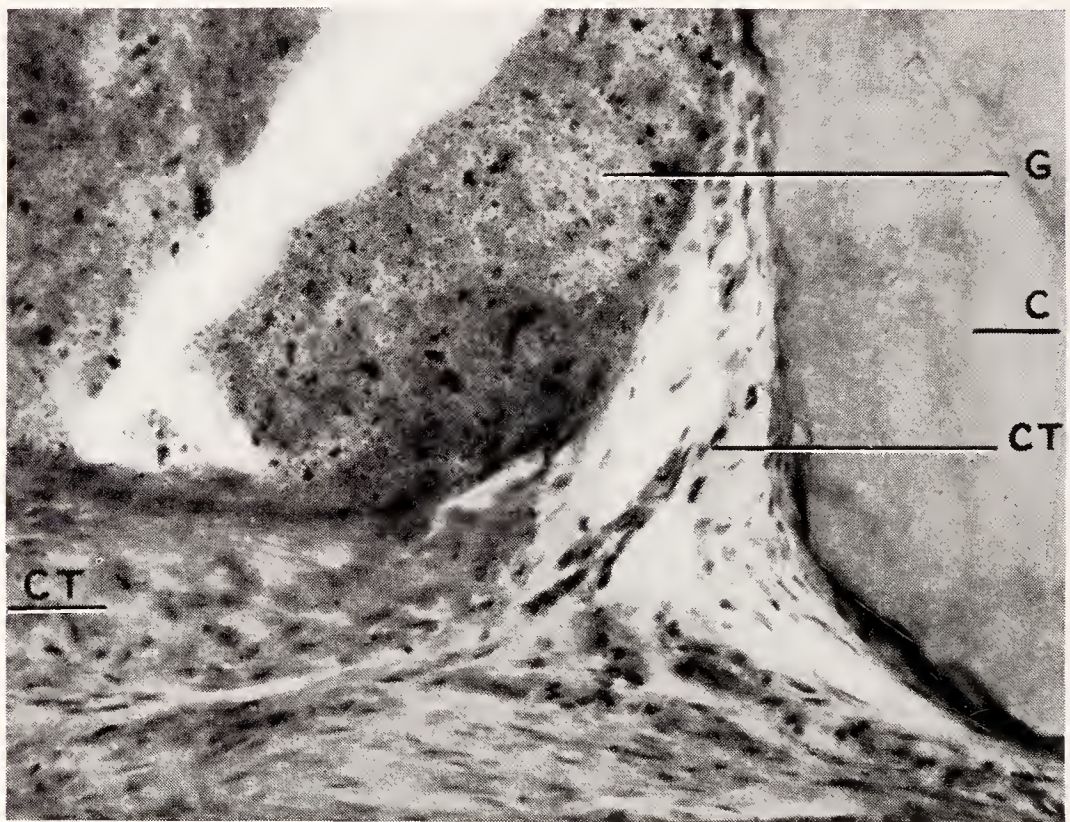


FIG. 210.—High magnification of Figure 209. *G*, guttapercha; *C*, cementum; *CT*, fibrous connective tissue of periodontal membrane lying in direct contact with guttapercha and wall of root canal. Notice the absence of any inflammatory reaction in the periapical connective tissue. (Coolidge, Jour. Am. Dent. Assn.)

Tissue Changes Following Root Resection.—Similar changes may take place after a root resection has been performed. Large areas of bone destruction around the apex of an infected, pulpless tooth may heal completely after the root end has been resected and the pulp canal filled. Several such teeth have been examined microscopically (Aisenberg, Blayney, Blum, Coolidge, Hill). They showed a normal periodontal membrane without inflammatory tissue changes, and deposition of cementum upon the resected dentin surface. The case published by Coolidge will be illustrated here. Figure 211, *A*, shows an upper first bicuspid; this radiograph was taken in 1914, immediately preceding the root resection. Fourteen years later, in 1928, the bone had been completely regenerated (Fig. 211, *B*). The histological examination of the root end revealed

the presence of a continuous layer of newly formed cementum upon the dentin surface (Fig. 211, *C'*); the end of the guttapercha cone is covered by fibrous connective tissue without any evidence of inflammation (Fig. 212). The periodontal tissue extending from the newly formed cementum on the root end to the bone has the typical fibrous structure of a normal periodontal membrane.

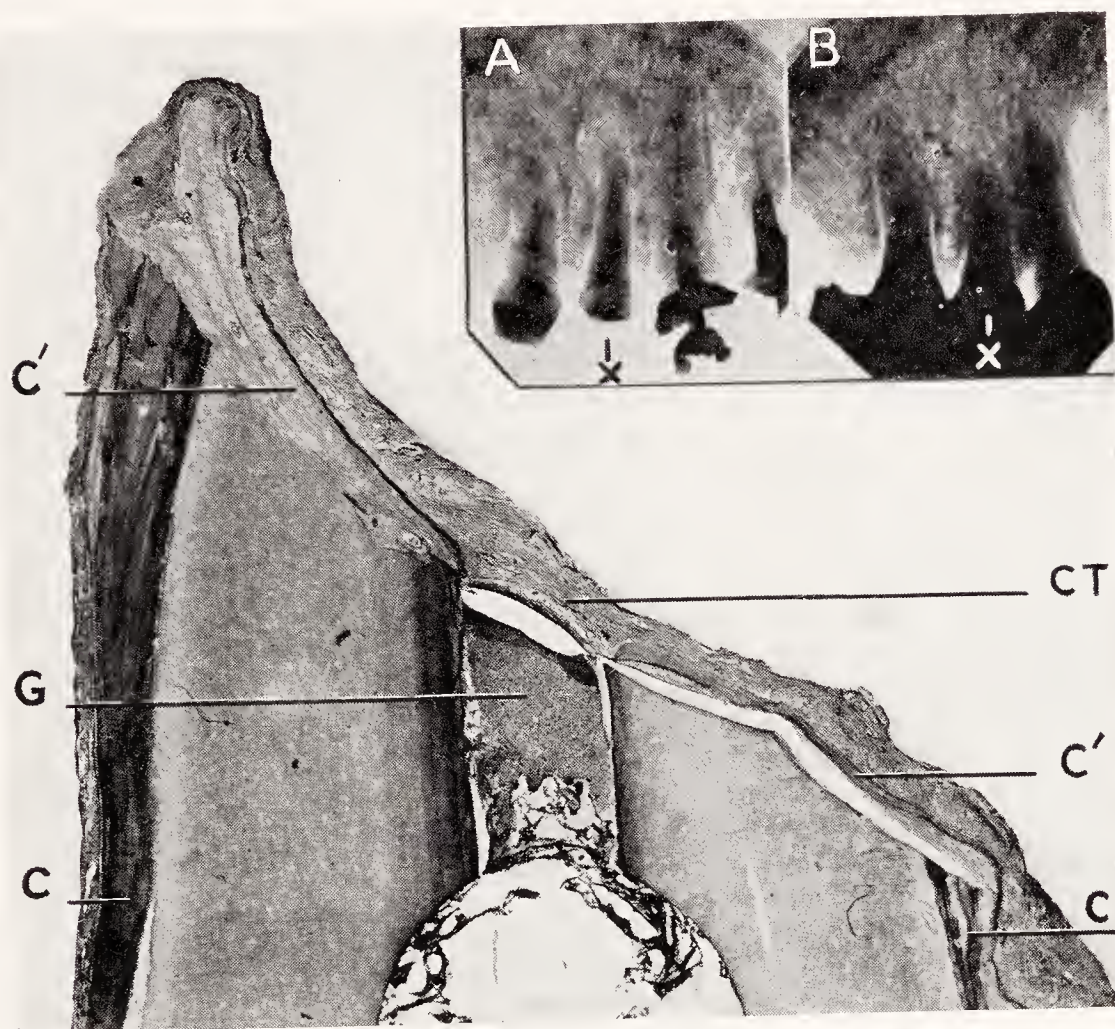


FIG. 211.—Tissue repair following root resection. *A*, radiograph of an upper second bicuspid with imperfect root canal filling and marked destruction of the periapical bone. This radiograph was taken in 1914, immediately before the root resection was performed. *B*, same tooth as it appeared in 1928. The area formerly occupied by the apex and the granuloma is entirely filled by bone. Bucco-lingual section through resected root end. Deposition of cementum and regeneration of a normal periodontal membrane on the resected surface. *G*, guttapercha root canal filling; *C*, original cementum on root surface; *C'*, cementum deposited on the resected root surface; *CT*, connective tissue capsule bridging the root filling material. (Coolidge, Jour. Am. Dent. Assn.)

The deposition of cementum upon the dentin of a formerly undoubtedly infected root and the regeneration of a normal periodontal membrane are of great practical importance; they show that complete healing in such a tooth is actually possible. The process of healing following infection of root canal and periapical tissues has been studied experimentally by Stein and by Dixon and Rickert, who used the teeth of dogs for their investigations. The teeth were infected with pathogenic microorganisms (streptococci). Then the teeth were sealed and the animals kept under observation for several

months. During this time, the development of an area of bone destruction around the apex could be observed radiographically. The infection spread beyond the foramen and involved the bone. Then the root canals were opened under aseptic conditions and treated in the same manner they would have been under identical circumstances in man. After the canals had been filled, the animals were permitted to live for another period of time, in some cases as long as eighteen months. During this time, radiographic check-ups revealed that the areas of bone destruction around the apices were decreasing in size and in some instances had disappeared entirely. The animals were then killed, and the treated teeth together with

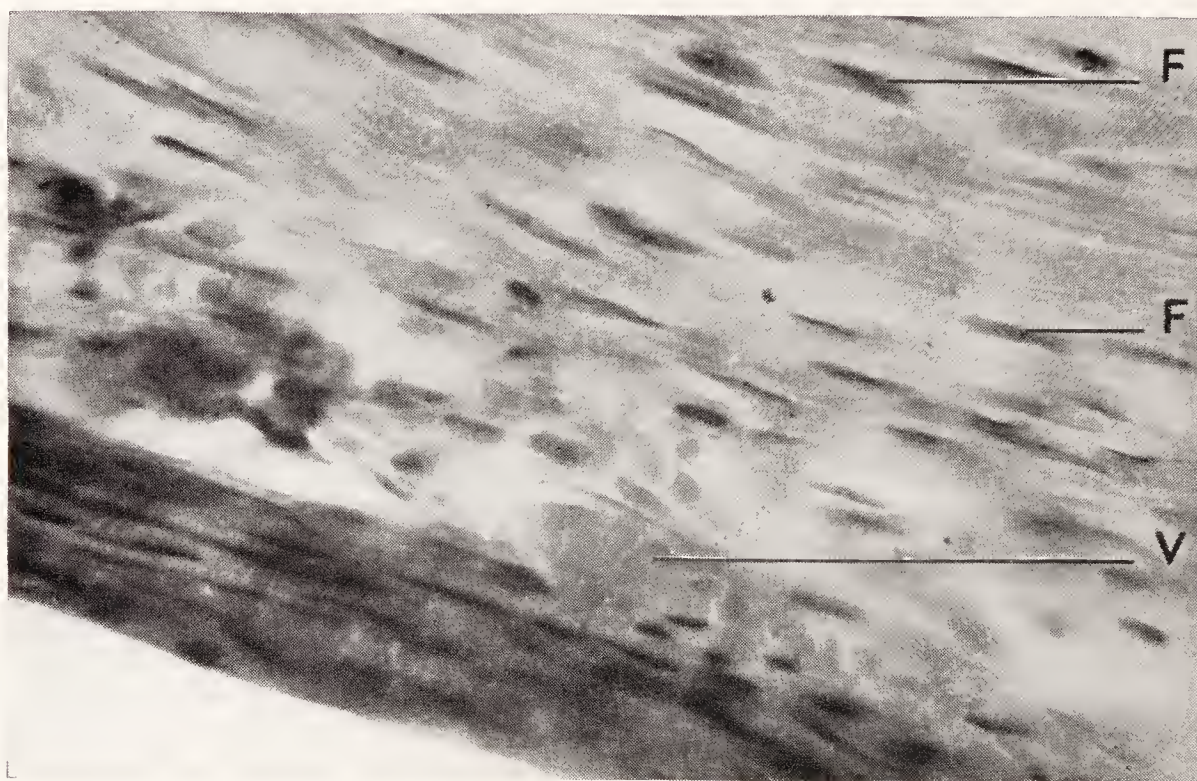


FIG. 212.—High magnification of the connective tissue covering the root canal filling in Figure 211. *F*, fibroblasts; *V*, capillary vessel containing red blood corpuscles. (Coolidge, Jour. Am. Dent. Assn.)

the surrounding jaw tissues were sectioned. Thus, it was found that such areas of chronic inflammation may heal: new bone is built, scar tissue forms on the surface of the root and over the root filling material, the apical foramina are sealed by cementum, and the periodontal membrane is regenerated. The histological findings in these infected and treated animal teeth are identical with those found in human teeth with a similar clinical and radiographic history.

In summarizing, it must be said that the prognosis of pulpless, infected teeth must still be considered doubtful. However, in view of the fact that evidence of complete healing and repair following treatment of such teeth has been produced both clinically and microscopically, attempts to save such teeth seem to be well justified.

As the methods and technique of root canal therapy improve and develop, the number of pulpless, infected teeth that can be saved will no doubt increase.

PULP AMPUTATION.

Pulp amputation is an operation in which only the coronal portion of the pulp is removed, while the root portion is left undisturbed in the pulp canal. This is done in order to save teeth with exposed or diseased, but vital, pulps; it cannot be used if the pulp is necrotic. In several European countries pulp amputation is the method of choice for the treatment of diseased pulps. Its main advantages are the relative ease with which it can be done and the avoidance of all the difficulties caused by the anatomical variations of the human root canals. This operation has never been popular in the United States, and only during the past ten or twelve years has there been any special interest shown in it.

Pulp amputation can be performed by either of two fundamental methods. In the one method, the pulp is devitalized by arsenic trioxide, the crown portion removed, and the devitalized pulp stump covered with a mummifying paste containing formocresol; in the second method, the pulp is anesthetized by infiltration anesthesia, the crown portion removed, and the living pulp stump covered with a non-irritating material under which healing of the pulp tissue takes place. The tissue changes associated with these two methods will be discussed separately.

Pulp Amputation With Mummifying Paste.—The amputation of the pulp following devitalization with arsenic trioxide is extensively used in Switzerland and Germany. The technique is fairly simple: Arsenic is applied to the pulp for forty-eight hours. Then under rubber dam and rigid aseptic conditions, the coronal portion of the pulp is removed with large round burs, and the pulp chamber filled with a paste consisting of formocresol in an inert vehicle, such as zinc oxide. Hess and other Swiss investigators use Gysi's Trio-paste; in Germany a similar paste is extensively used that was worked out by the author's father, Dr. Robert Kronfeld. The mummifying paste is covered with cement, and the latter by a permanent filling.

The tissue changes following this operation consist of a gradual resorption and replacement of the mummified pulp tissue in the canal by ingrowing periodontal connective tissue; subsequently this ingrowing tissue produces cementum that reduces the lumen of the root canal and eventually completely obliterates the canal. These changes have been illustrated in American literature by Hess and

Bossard. One of Hess' illustrations (Fig. 213) shows the apical portion of one of the roots of a lower molar four years after the pulp had been devitalized with arsenic and the pulp stumps mummified with Gysi's Triopaste. The lumen of the canal is greatly reduced by an extensive deposition of cementum that extends crownward to the level of the mummified pulp.



FIG. 213.—Longitudinal section through lower first molar root; boy, fifteen years. Pulp amputation and mummification with Gysi's Triopaste four years before extraction. The root canal had been enlarged by resorption and then was filled with secondary cementum up to the level of the mummified pulp. (Hess, *Dent. Items Int.*)

An earlier stage of replacement of an amputated pulp stump by ingrowing connective tissue and cementum is shown in Figure 214. This work, done by Rzeszotarski, confirms what has been found by the Swiss investigators and furnishes evidence that correctly performed pulp amputation with formocresol is a biologically sound method of treatment.

Amputation of the Vital Pulp.—The amputation of the coronal portion of the pulp without previous devitalization is performed under infiltration anesthesia. The remaining pulp stump is covered by a material that permits healing of the amputated living pulp surface. This operation is also known as pulpotomy or partial

pulpectomy. Various drugs have been used for this purpose, all with good results both clinically and histologically. This method has been particularly successful as the treatment for pulp exposure in young teeth with incompletely formed roots. In such teeth, as a rule, the usual method of pulp removal and root canal filling gives unsatisfactory results. If, however, only the coronal portion of the pulp is removed and the root portion is kept vital, root formation

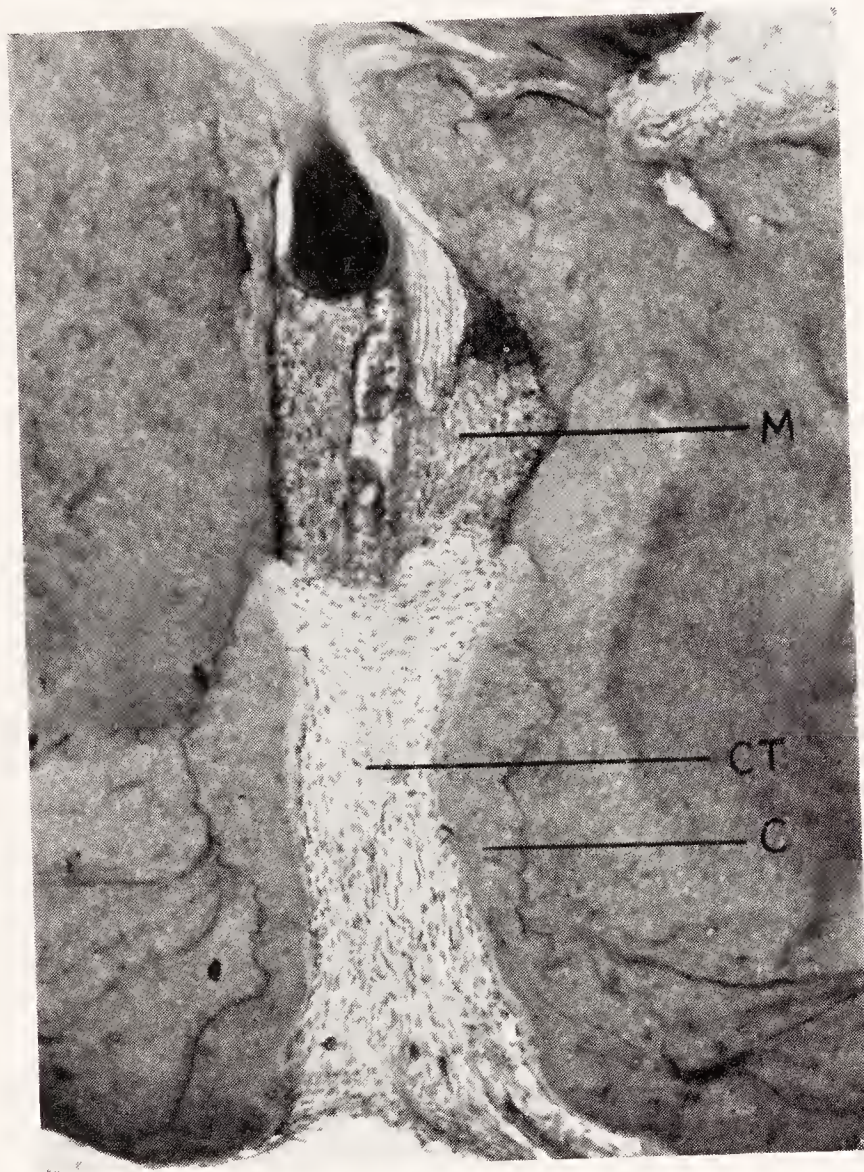


FIG. 214.—Root end of lower cuspid the pulp of which was devitalized by arsenic, amputated, and mummified with Gysi's Triopaste. *M*, mummified pulp tissue; *CT*, ingrowing periodontal connective tissue replacing the mummified pulp; *C*, cementum deposition along the walls of the root canal. (Courtesy of Joseph S. Rzeszotarski.)

continues, and eventually the radiograph shows a normally formed root end. Cementum deposition inside of the root canal tends to reduce the lumen and eventually obliterates the canal.

The extensive experimental work of Hellner, Gottlieb, Orban and Stein, and others has shown that amputation of a vital pulp is particularly successful if dentin splinters are implanted into the tissue of the pulp stump during the operation. Such small particles of dentin act as nuclei around which calcification takes place, which finally completely seals the pulp. Figure 215 shows a specimen of

this kind. The coronal portion of the pulp of this tooth, an upper bicuspid, had been removed under infiltration anesthesia and the pulp chamber filled with a mixture of zinc oxide and eugenol. The tooth was extracted two months and fifteen days after the operation. Beneath the amputation paste, a bridge of calcified tissue has formed

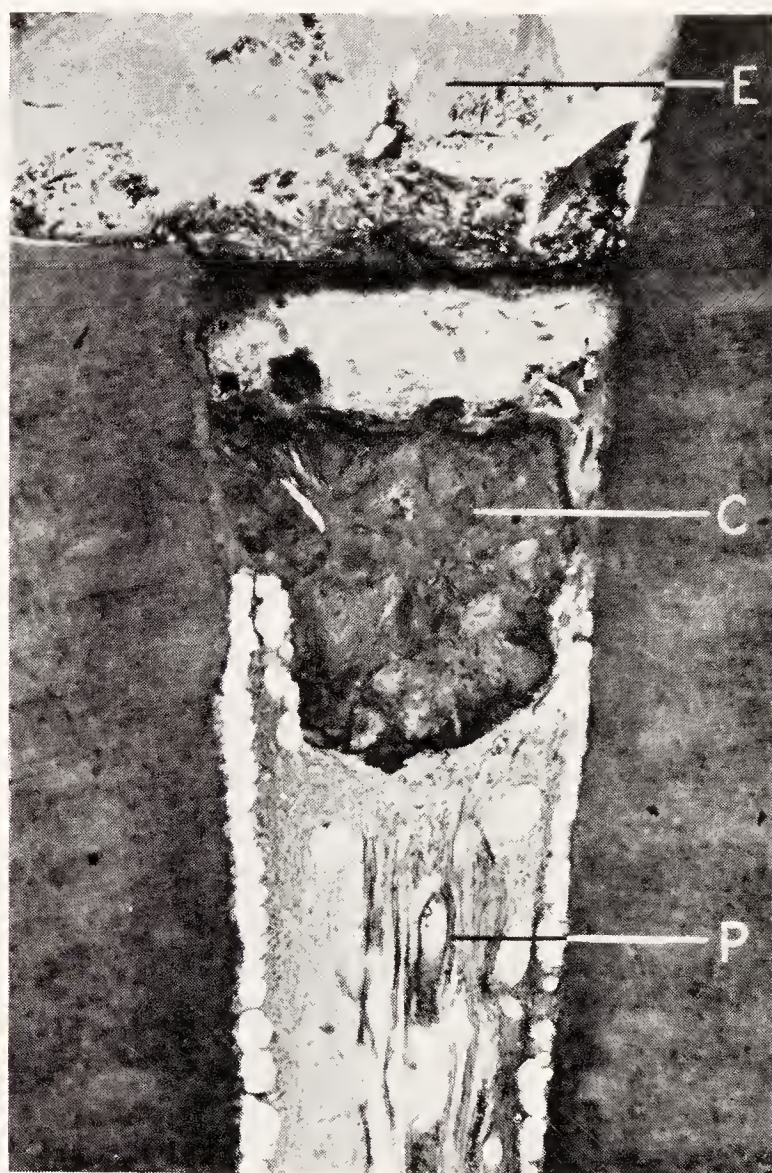


FIG. 215.—Amputation of a vital pulp in an upper bicuspid. The tissue from the pulp chamber was removed under infiltration anesthesia and the pulp stumps covered with a paste of zinc oxide and eugenol. *E*, paste in pulp chamber; *C*, plug of calcified material sealing the pulp stump; *P*, pulp stump free of inflammatory changes. (Courtesy of Joseph S. Rzeszutarski.)

that almost completely closes the root canal. Only in one area is there a small opening alongside this calcified barrier. A higher magnification reveals that the calcified material consists of irregular dentin laid down around small splinters of dentin that the bur had torn loose from the walls of the pulp chamber during the operation. Beneath the calcified tissue the pulp is histologically normal.

Needless to say, any amputation will fail unless a rigid aseptic technique is followed throughout the entire operation. The introduction of microorganisms into the field of operation invariably leads to decomposition of the pulp stump and infection of the apical periodontal tissues.

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CHAPTER X.

BIOLOGY OF CEMENTUM.

DEVELOPMENT AND STRUCTURE OF CEMENTUM.

CEMENTUM is a calcified tissue that covers the surface of the root. It is a product of the connective tissue of the periodontal membrane, consisting of an organic ground substance (matrix) into which inorganic salts are precipitated. The deposition of cementum begins while the root portion of the tooth germ is developing within the jaw; under physiological conditions it continues throughout life, so that the thickness of the cementum increases steadily as the tooth grows older.

In order to understand the various structural changes found in cementum at different ages, it is necessary first to consider its development. Sections stained with hematoxylin and eosin do not show the fine details of early cementum formation; therefore, it is necessary to study sections stained with silver. Then it is possible to see the first delicate layers of cementum and their relationship to the collagenous fibers of the periodontal membrane.

After the dentin of the root surface has been formed, it lies in contact with the connective tissue that surrounds the germ. The functional connection between the dentin and the connective tissue is later established by a layer of cementum that is deposited upon the dentin surface. During this deposition collagenous fibers of the adjacent connective tissue are embedded into the forming cementum, establishing the first true connection between tooth and periodontium. Since the other ends of these fibers are attached to the bone, the foundation for later functional connection between cementum, fibers, and bone is laid.

When the tooth erupts the cementum has already attained a certain thickness (Fig. 216). The fiber bundles, the ends slightly thickened, extend only to the surface of the cementum. Their course, however, is decidedly different from that found in unerupted teeth; they are arranged in bundles, as is characteristic of the principal fibers of functioning teeth, and run at an angle of about 45 degrees from the root surface to the alveolar bone. The cementum has only a fine fibrillar structure, no continuation of the fiber bundles into the cementum being visible.

In the study of the morphology of the cementum it is usual to differentiate between primary or cell-free cementum and secondary or cell-containing cementum.

Primary cementum contains no cells and shows only a fine fibrillated pattern at right angles to the root surface (Fig. 217). It is usually found in the coronal portion of the root. Primary cementum rarely becomes very thick.

Secondary cementum contains cells embedded in the matrix, which give it an appearance very much like bone. It is usually found in the apical portion of the tooth; its extension crownward varies

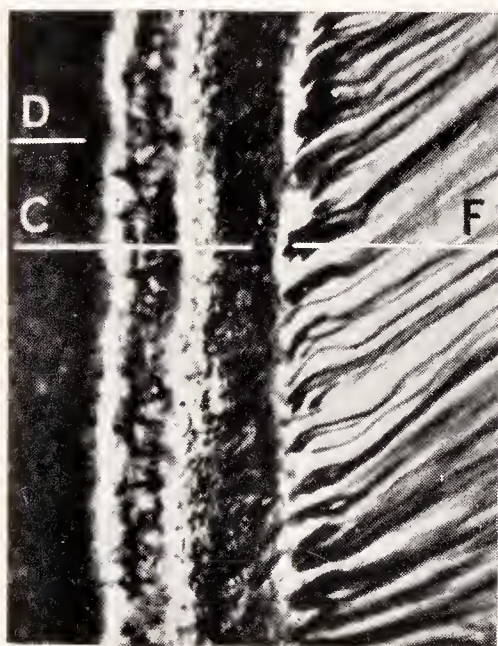


FIG. 216

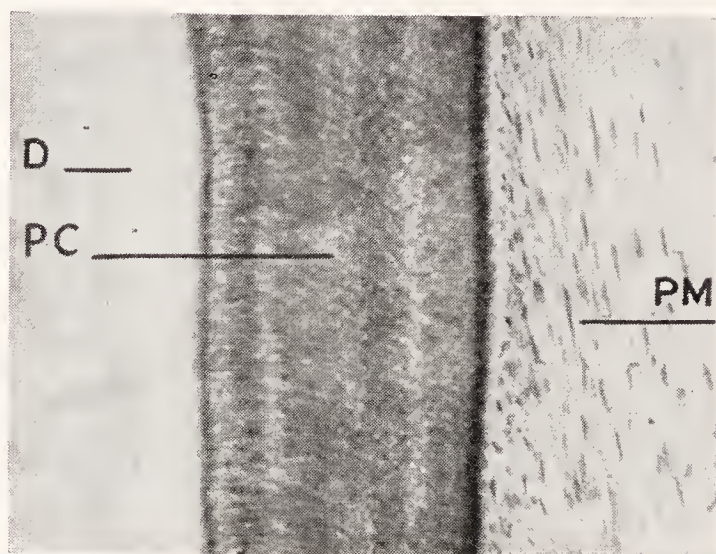


FIG. 217

FIG. 216.—Cementum in an erupting molar of a child. The fibers of the periodontal membrane are attached only to the superficial layers of cementum. No continuation of these fibers into deeper layers of cementum is visible (Bielschowsky stain). *D*, dentin; *C*, cementum; *F*, attachment of the fibers to the surface of the cementum. (Kronfeld, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

FIG. 217.—Primary (cell-free) cementum. *D*, dentin; *PC*, primary cementum showing a fine fibrillar structure; *PM*, periodontal membrane.

in different teeth. Contrary to statements occasionally encountered in dental literature, there is no functional difference between primary and secondary cementum. It is not yet known why in some teeth the major portion of the root surface is covered by primary cementum, while in others secondary cementum is predominant, but as far as functional and biological significance are concerned, the two types of cementum are identical. As will be shown later (Figs. 222, 223, and 224), a resorbed area on the root surface may be repaired either by primary or by secondary cementum, with apparently no difference in the functional value of the repaired area.

Secondary cementum contains cementum corpuscles, cementum cells or cementocytes, which lie in lacunæ in the matrix. These cells,

which are derived from the periodontal membrane, are cementoblasts that have become embedded in the matrix. Each lacuna in the secondary cementum is connected with neighboring lacunæ by fine channels, the canaliculi, which contain the protoplasmic processes of the cells in the lacunæ (Fig. 218). Thus, a system of channels is established by which fluids may pass from the periodontal membrane into the superficial layer of cementum and from there to deeper strata. The presence and arrangement of the cells make the secondary cementum appear similar to bone; hence the name osteocementum is sometimes used for this hard tissue.

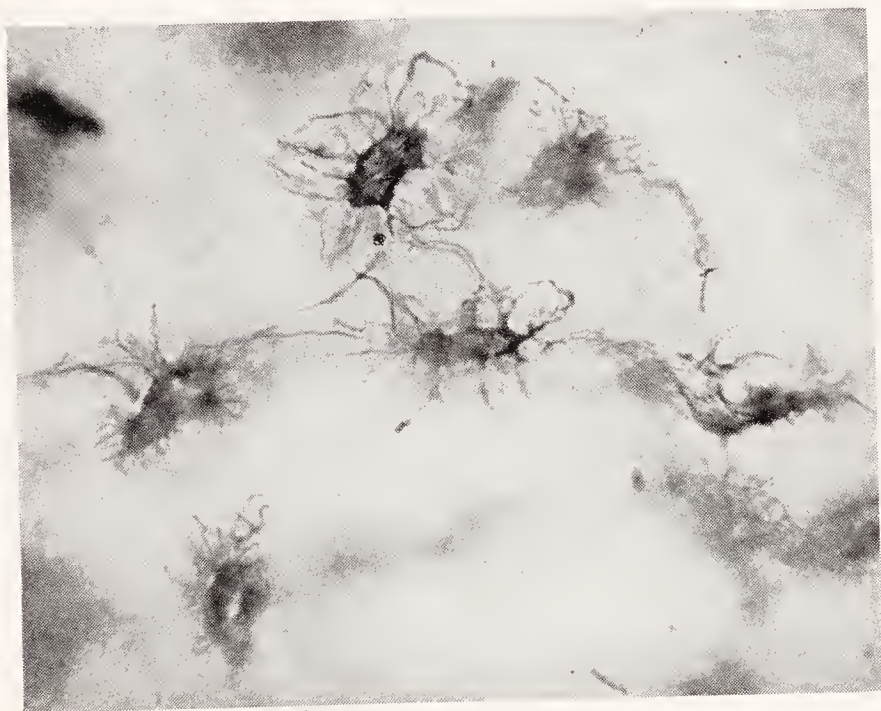


FIG. 218.—Lacunæ and canaliculi in secondary cementum (stained ground section). The canaliculi radiate from each lacuna and are connected with canaliculi from the neighboring lacunæ.

Secondary cementum usually shows a distinctly lamellated structure. The lamellæ run parallel to the root surface; they are the layers of consecutive deposition and calcification. The attachment of the fiber bundles of the periodontal membrane to the secondary cementum is analogous to their attachment to the primary cementum, the fibers extending only as far as the most superficial layer of cementum. Only in cementum hyperplasias is it sometimes possible to demonstrate the continuation of the fiber bundles into the deeper layers of the secondary cementum (Fig. 219).

It is difficult to give any definite data on the distribution of primary and secondary cementum on the root surface. It has been found that most human permanent molars shortly before eruption have very thin layers of primary cementum on the surfaces of the roots; in addition the sides of the roots that face the interradicular septum and the bifurcation are covered with thick layers of secondary

cementum. Since these teeth are still completely embedded in the child's jaw, function cannot be held responsible for the variations in the distribution of cementum on different parts of the root surface.

It may be advisable to compare cementum and bone, and to correlate the physiological changes that take place during life in both of these hard tissues.

Morphology.—Bone has a calcified matrix that is arranged in lamellæ, either running parallel to the bone surface or having a circular arrangement around a central lumen. In the matrix are spaces (lacunæ) containing bone cells. These cells are connected

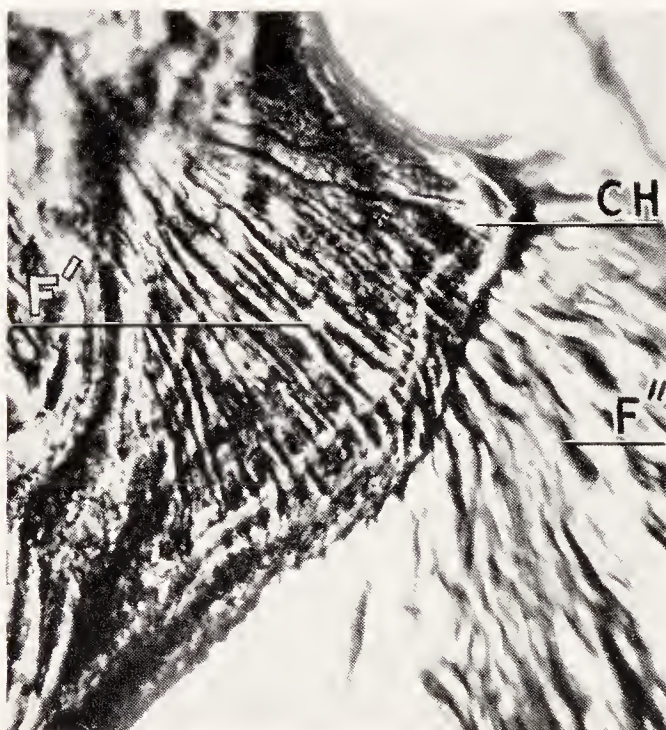


FIG. 219.—Sharpey's fibers embedded in a small cementum hyperplasia. The fibers can be traced into the deeper layers of cementum (Bielschowsky stain). CH, cementum hyperplasia; F' , Sharpey's fibers embedded in the cementum; F'' , principal fibers of the periodontal membrane. (Kronfeld, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

with each other by fine, protoplasmic extensions lying in the bone canaliculi.

Cementum also has a calcified matrix, identical to bone matrix, which is arranged in lamellæ parallel to the root surface. Primary cementum is free of cells. Secondary cementum has lacunæ that contain cementum cells, which are similar to bone cells and which likewise are connected to each other by canaliculi.

Physiology.—In bone a continuous process of elimination by resorption and replacement by newly formed bone takes place; thus, the total amount of adult bone remains fairly constant.

In cementum, areas of less vitality are not resorbed but are covered by a new, vital layer of cementum. Thus, the total amount of

cementum increases steadily throughout life; under physiological conditions no resorption occurs.

FUNCTION OF CEMENTUM.

In the preceding pages it was shown that the fiber bundles of the periodontal membrane enter only the superficial layers of the cementum; no fibers are visible in the deeper layers. Among dental histologists, Gottlieb was one of the first to call attention to the biological importance of cementum and to make this hard tissue the subject of intensive study. He thought of it as "protective cementum" (Schutzzement), as a defense mechanism of the organism against any conditions that might weaken the support of the tooth. Gottlieb expressed the following opinion concerning the physiological changes in the cementum: the fiber bundles of the periodontal membrane, like any other tissue in the organism, are subject to involutionary changes; they live and function for a certain length of time, and then they die. Concurrently with this process of aging or involution, new fibers are built that take over the function of the old ones; these new fibers are embedded in a layer of new cementum. Thus, the continuous deposition of cementum throughout life can be regarded as a process by which calcified strata of cementum with low vitality are covered by new layers with new fiber attachments and high vitality.

That this conception of the change in vitality is more than a mere hypothesis can be demonstrated by a study of the cementum cells in the secondary cementum. These cells are indicators of the vitality of the surrounding cementum. In the root of a healthy human tooth, the cementum cells in the superficial layer appear normal and well stained (Fig. 220). In a slightly deeper level the cells show degenerative changes; the nuclei are small, the cell bodies shrunken and irregular. Still deeper in the cementum the lacunæ are empty, the cells having completely degenerated and disappeared. Figure 221 is a higher magnification of cementum cells from the different layers in Figure 220. In Figure 221, *A*, which was taken of an area of cementum near the surface, two young cells are seen. The cell bodies are slightly shrunken away from the walls of the lacunæ; otherwise, they do not show any pathological changes. In a slightly deeper layer (Fig. 221, *B*), the shrinkage of the cells is much more pronounced; a large portion of the lacuna appears empty. Still farther away from the surface the nuclei of the cementum cells have disintegrated; remnants of the nucleus and fat granules are

found in the lacuna and in some of the surrounding canaliculi (Fig. 221, *C*). Finally, in the deepest layer of cementum, the cells have completely disappeared; the lacunæ are empty except for some

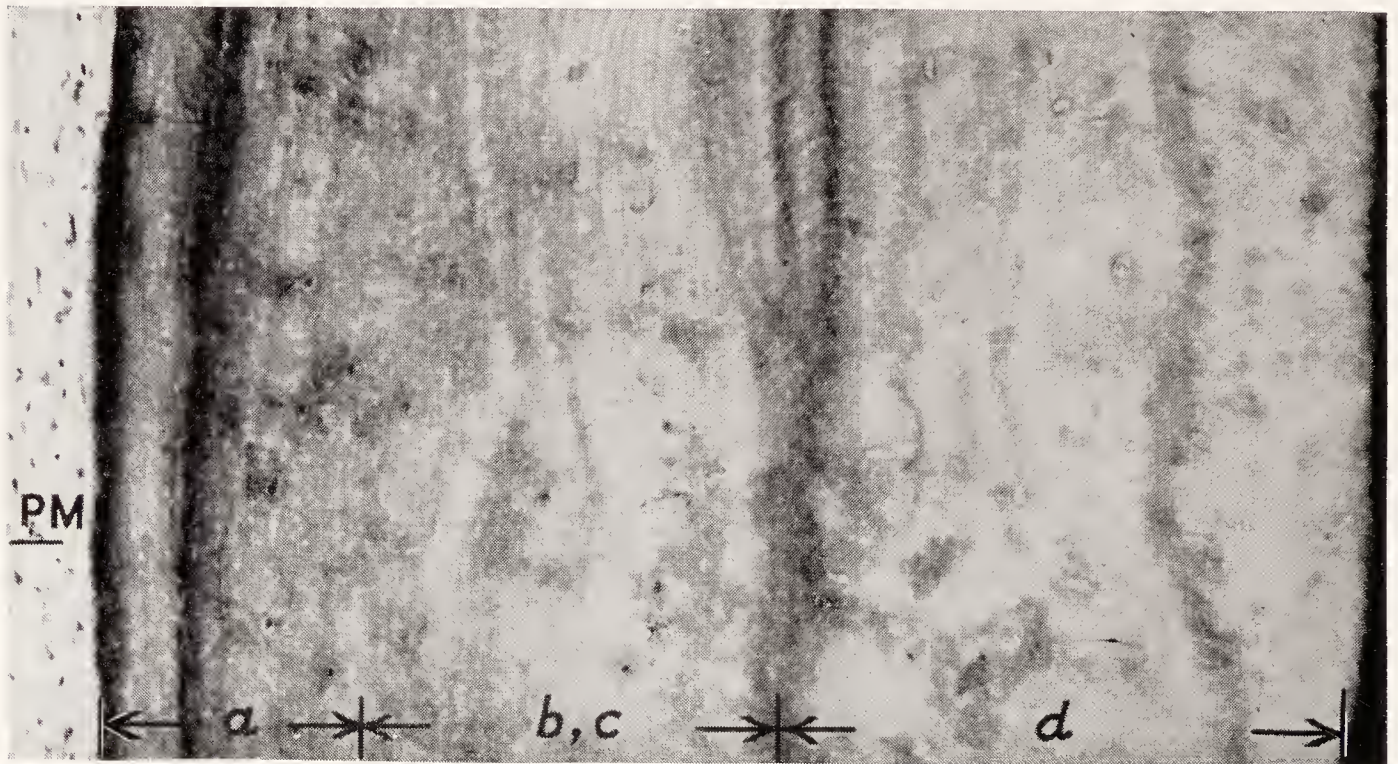


FIG. 220.—Thick secondary cementum on the root of an intact upper bicuspid. *PM*, periodontal membrane. *a*, area of vital cells. The cementum corpuscles in this area are well stained and appear normal. *b, c*, area of degenerating cells; *d*, area of dead cells. All lacunæ of the cementum are empty.

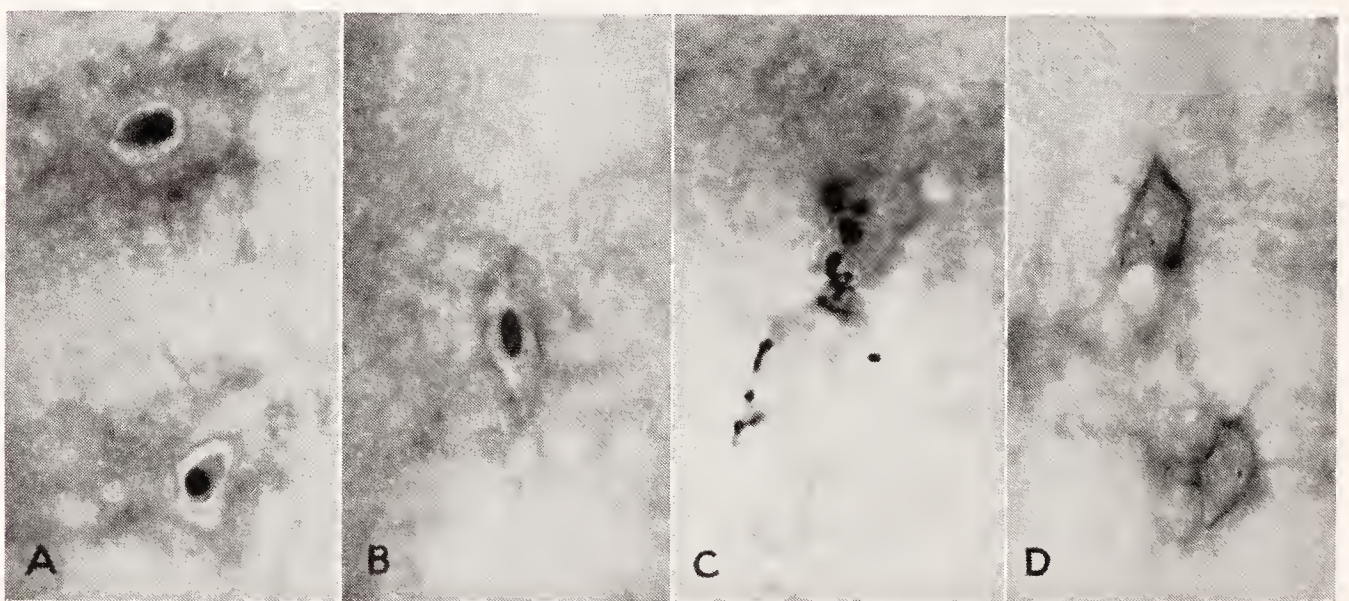


FIG. 221.—High magnification of cementum corpuscles in different layers of Figure 220. *A*, near the surface of the cementum (Fig. 220, *a*): normal cementum cells showing nucleus and cell body; *B*, slightly deeper (Fig. 220, *b*): cementum cells are beginning to shrink; *C*, still deeper layer (Fig. 220, *c*): disintegration of cell; droplets of fat and protoplasm in lacuna and canaliculi; *D*, in the deepest layers of cementum (Fig. 220, *d*): lacunæ containing cell debris.

fine, dust-like debris (Fig. 221, *D*). Thus the changing condition of the cells in various depths indicates the high vitality of the young, superficial layers of cementum, and the low vitality of the deeper

layers. The oldest cementum in the deepest strata has lost its vitality; it acts merely as mechanical support.

In cell-free cementum the vitality changes cannot be demonstrated by changes in the cells; advancing calcification and concurrent biological deterioration are revealed by the progressive calcification and disappearance of the embedded fibers of the periodontal membrane.

The space necessary for the deposition of cementum on the root surface is created by the minute bone resorptions constantly occurring in the socket of every functioning tooth. Slight excess of functional stress exerted upon the crown causes resorption of the alveolar bone; the result is a widening of the periodontal space, which is partly compensated for by the formation of new cementum. Another way in which space is created for cementum deposition is by the gradual occlusal movement of all teeth throughout life. This movement results in a widening of the periodontal space, especially at the apex and in the bifurcation in case of multi-rooted teeth, and this space in turn is filled with cementum, which reduces the periodontal space to its normal width. Cementum formation as compensation for the occlusal movement of a tooth was observed as far back as 1909, when Loos called attention to the relative thickness of the cementum of extruding teeth without antagonists. The best way to account for the cementum deposits in such teeth is to consider them a reparative or defensive measure against rapid occlusal displacement.

The rôle of cementum in repairing any damage or injury to which a root may be subjected is of great practical importance. Cementum covers injured areas, reestablishes function, and reunites parts that were separated by a trauma (fracture).

One of the common forms of injury to the root surface is shallow resorptions caused by excessive occlusal stress. Such resorptions are usually encountered in the teeth of individuals who use their teeth forcefully; such people grind their teeth or bite on hard objects with such force as to cause damage to the periodontal membrane and produce circumscribed resorption of the root surface. These small damages are invariably repaired after the injured periodontal membrane has recovered. New cementum is deposited upon the original cementum or dentin in the resorbed area; new Sharpey's fibers are embedded into this cementum, and thus the damaged portion of the root surface is restored to its normal functional properties. The reparative cementum may be either primary or secondary, or both. In resorptions in the apical portion of the root, secondary cementum will most commonly be found; in the coronal

portion of the root, either primary or secondary cementum may be deposited. Many times, primary cementum is deposited directly upon the resorbed root surface (Fig. 222). Other times, cell-containing secondary cementum is deposited first, and the regenerated fiber bundles of the periodontal membrane become embedded in the

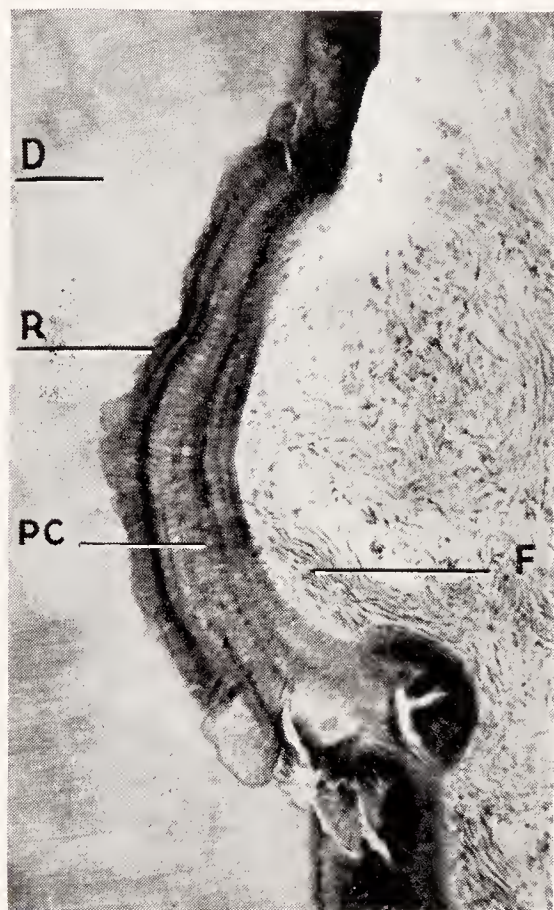


FIG. 222.—Repaired resorption of the root surface. The reparative cementum is primary cementum. *D*, dentin; *R*, line of resorption in the dentin; *PC*, primary cementum in the resorbed area; *F*, periodontal fibers embedded in the regenerated cementum surface.

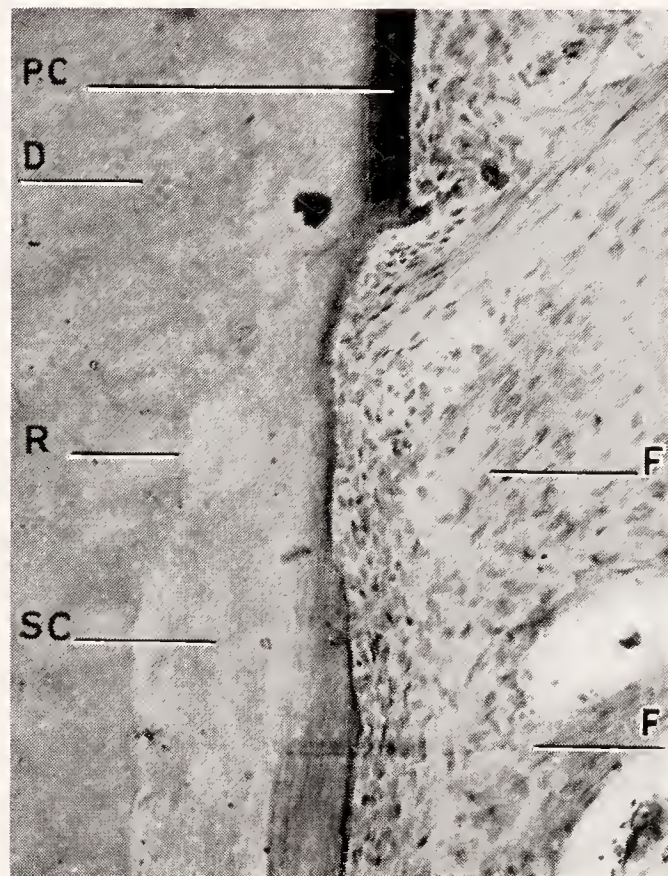


FIG. 223.—Repaired resorption of the root surface. The reparative cementum is secondary cementum. *D*, dentin; *R*, line of resorption in the dentin; *PC*, primary cementum on the original root surface; *SC*, secondary cementum in the resorbed area; *F*, periodontal fibers embedded in the the secondary cementum.

reparative cementum (Fig. 223). Again secondary cementum is the first hard tissue to be formed in the resorbed area, a layer of primary cementum being deposited on top of it (Fig. 224). From the viewpoint of restoration of function in the damaged area, either form of repair seems to be satisfactory.

Another occasion for reparative cementum formation is injury to the root end in root canal therapy. Here, cell-containing secondary cementum is most frequently found. In the absence of infection, the periodontal membrane reacts to the removal of the pulp and to the insertion of a foreign material into the root canal by depositing cementum, which finally covers root filling material, pulp remnants, and ramifications as a solid callus and furthers the healing of the

apical wound. One interesting observation is the possibility of cementum deposition directly upon the surface of a foreign body, such as guttapercha; this condition must, in a biological sense, be looked upon as the ideal outcome and as the final goal in every pulp canal operation (see Chapter IX).

The rôle of cementum in tooth fractures will be discussed in detail in Chapter XVIII. If the continuity of the root has been severed

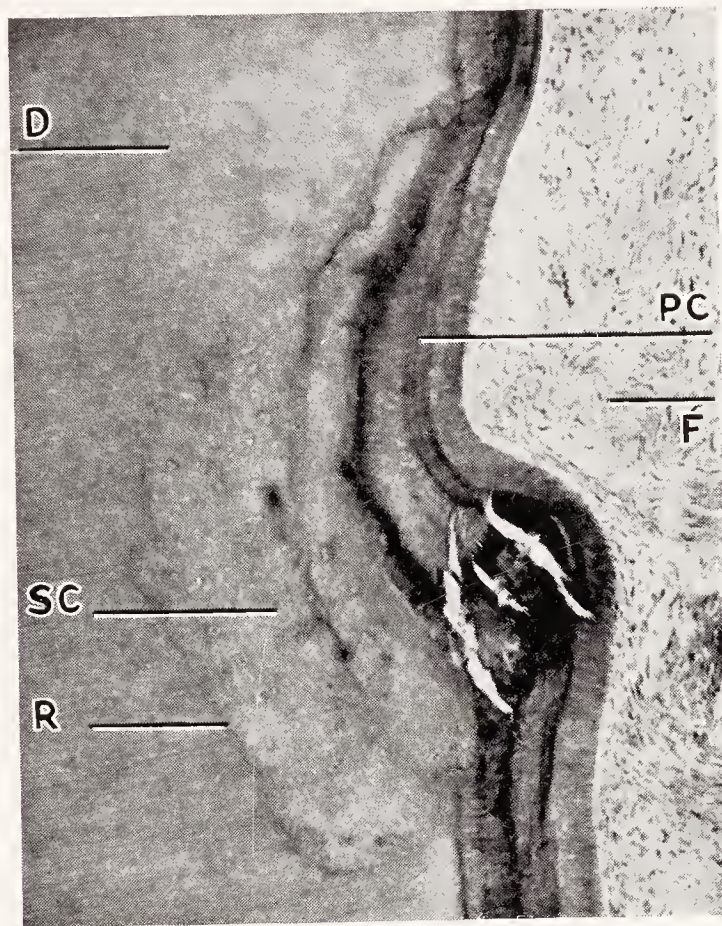


FIG. 224—Repaired resorption of the root surface. Repair first by secondary then by primary cementum. *D*, dentin; *R*, line of resorption in the dentin; *SC*, secondary cementum deposited upon the resorbed dentin surface; *PC*, primary cementum deposited on top of the secondary cementum; *F*, fiber bundles embedded in the primary cementum.

by a trauma, the periodontal membrane deposits cementum upon the fractured surfaces, and if the circumstances are favorable, the fragments may become united.

EXCESSIVE FORMATION OF CEMENTUM.

Abnormal or excessive formation of cementum occurs as cementicles, coronal cementum, and cementum hyperplasias.

Cementicles.—Cementicles are calcified bodies found in the periodontal membrane. They are usually small and seldom measure more than 0.1 to 0.2 mm. In the periodontal membrane of some teeth they are very numerous, in others entirely absent. The development of cementicles is closely associated with the epithelial rests in the periodontal membrane, and frequently epithelial cells can be observed

near the cementicles (Fig. 225). In these cases cementicles seem to form by the deposition of cementum around degenerating or dead epithelial cells (Gottlieb). The close relationship between cementicles and epithelium is also corroborated by the common

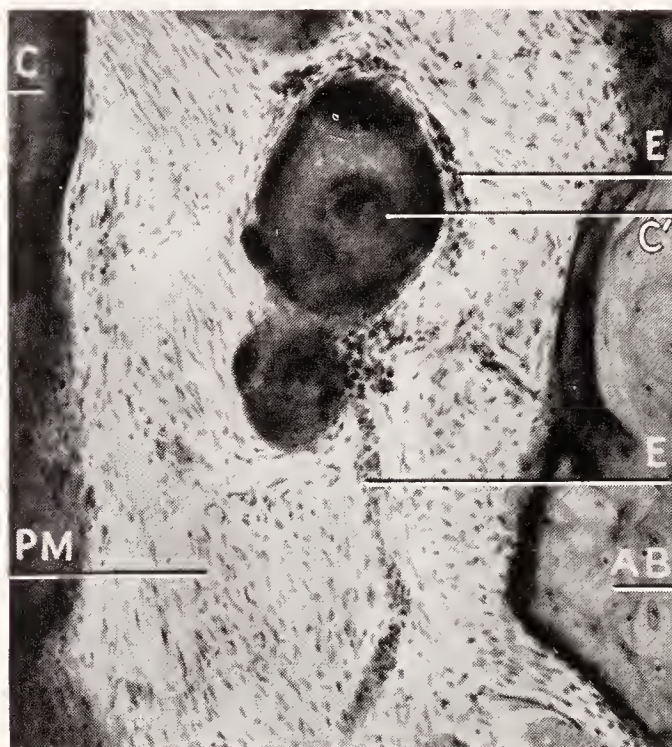


FIG. 225.—Cementicle in the periodontal membrane. *C*, cementum on the root surface; *PM*, periodontal membrane; *C'*, cementicle lying free in the periodontal membrane; *E*, epithelial strands near and around the cementicle; *AB*, alveolar bone.

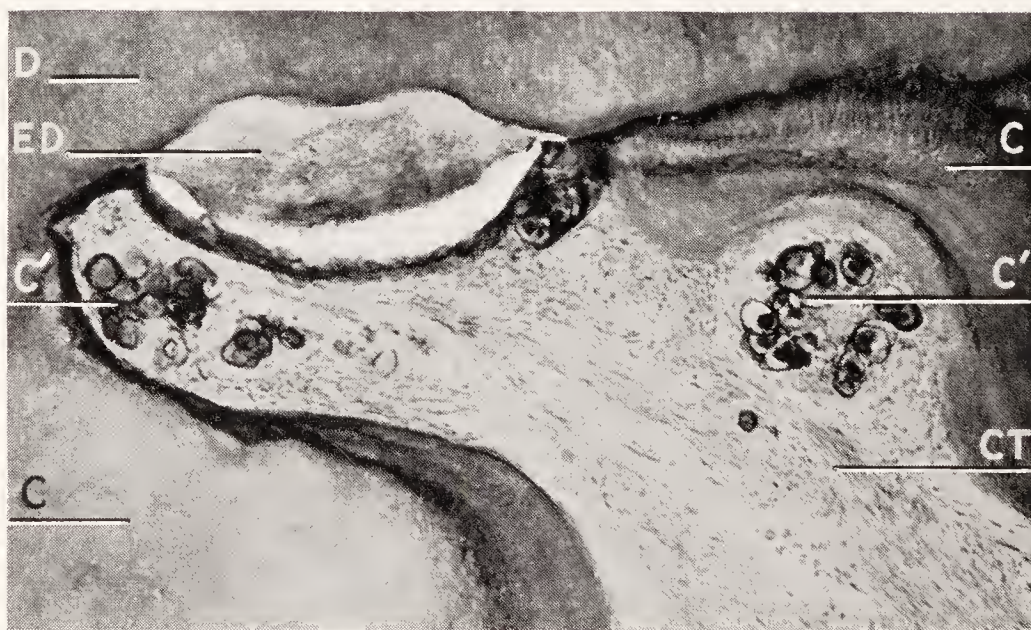


FIG. 226.—Cementicles in the proximity of an enamel drop in the bifurcation of a lower molar. *D*, dentin; *ED*, enamel drop; *C*, cementum; *CT*, fibrous connective tissue; *C'*, groups of small cementicles accumulated around degenerating epithelial cells.

occurrence of cementicles in the neighborhood of enamel drops. These atypical enamel formations are covered by a layer of epithelial cells which gradually degenerate and form centers of calcification around which cementicles develop (Fig. 226).

If cementicles are very numerous in the periodontal membrane of a tooth and lie close to the root surface, they may become adherent to the latter by the advancing formation of cementum, resulting in a peculiar, uneven appearance of the root surface (Fig. 227). Each one of the small prominences of cementum (cementum exostosis) shown in this illustration is a cementicle of concentric structure that originally lay free in the periodontal membrane and later became united with the root surface.

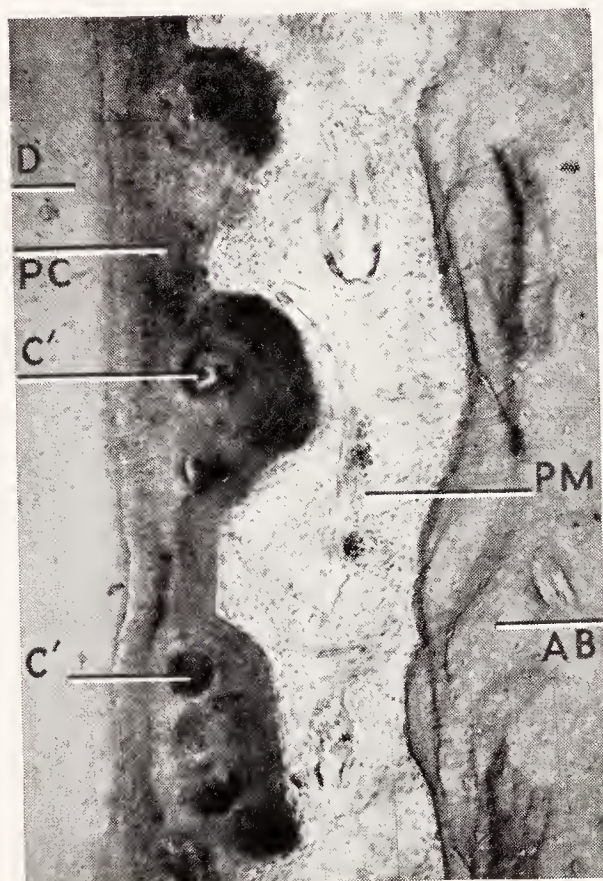


FIG. 227.—Adherent cementicles on the root surface of a lower molar. *D*, dentin; *PC*, primary cementum; *C'* cementicles of concentric lamellated structure embedded in primary cementum; *PM*, periodontal membrane; *AB*, alveolar bone.

Coronal Cementum.—Coronal cementum is cementum located on the enamel surface. In the herbivorous animals it normally covers almost the entire crown. In man it occurs as an anomaly.

In fully embedded teeth coronal cementum is not uncommon. As long as the enamel of an unerupted tooth is covered with enamel epithelium, no changes on its surface are possible. If, however, the epithelium becomes atrophic, the enamel comes in contact with the connective tissue of the tooth follicle, and cementum can then be deposited upon the enamel surface. Extensions of the cementum from the cemento-enamel junction over the crown are called cementum spurs; they are a rather common occurrence. Less frequently is cementum formed in the occlusal fissures of molars before eruption; if ground sections are prepared through such molars, it can be seen

that the fissures are filled with coronal cementum with its cementum lacunæ (Fig. 228).

Cementum Hyperplasia (Hypercementosis).—Cementum hyperplasia is an excess formation of cementum, either in a circumscribed area of the root or on its entire surface. The first type, circumscribed cementum hyperplasia, is very small, and is usually the result of calcification of the periodontal fiber bundles near their points of attachment. It is occasionally found on the surfaces of the roots

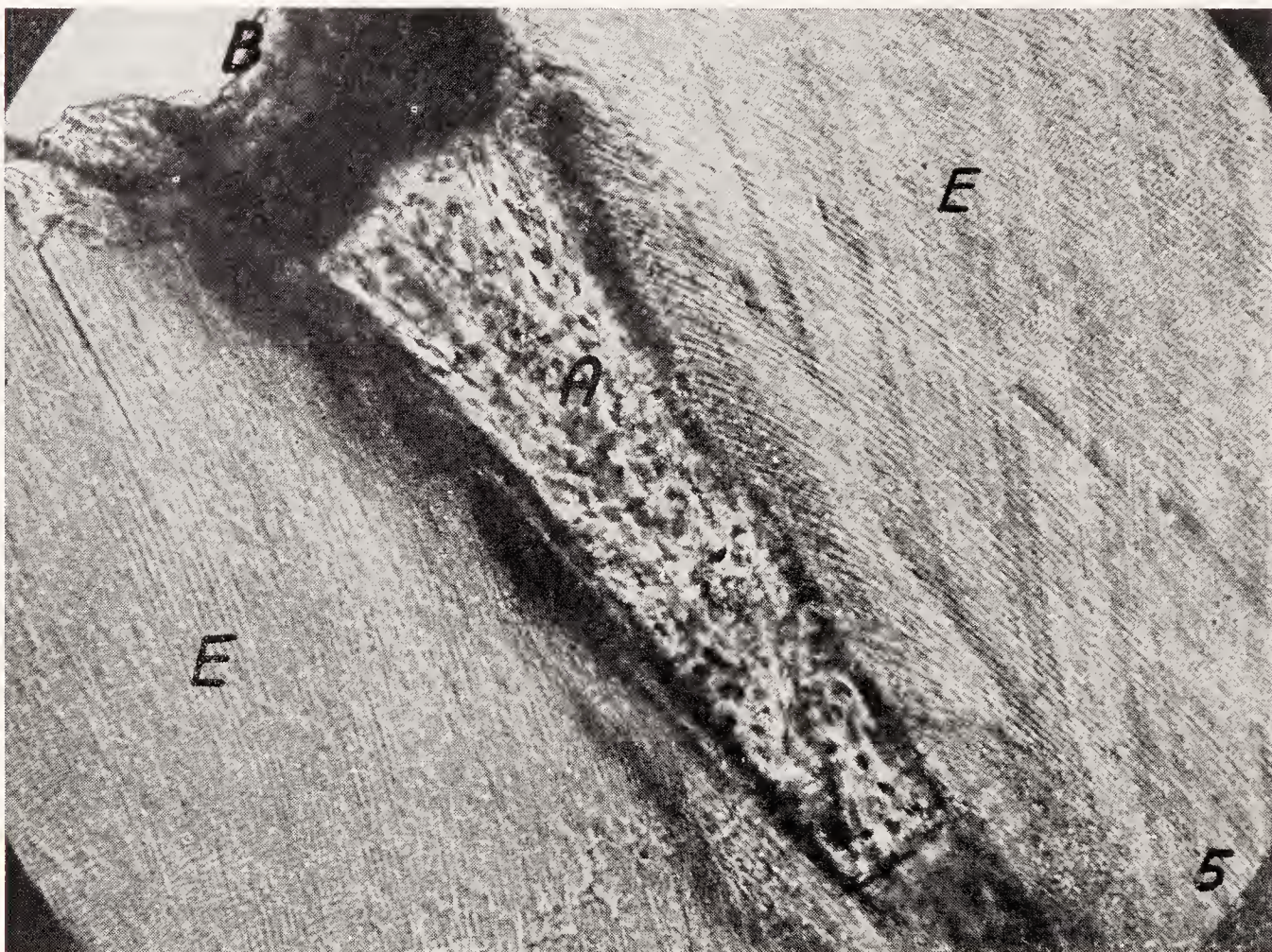


FIG. 228.—Ground section through an intact upper human molar. High magnification of occlusal fissure. *E*, enamel; *A*, coronal cementum with numerous cementum lacunæ; *B*, débris. (Kronfeld, Jour. Dent. Res.)

of teeth that are subjected to considerable functional stress; then the prominences or spike-like cementum hyperplasias to which the fiber bundles are attached may be compared to the bony projections or exostoses found at the point of attachment of tendons. Gottlieb found this type of cementum formation on the root ends of two lower incisors of an elderly man who used to allow a heavy pipe to rest on these two teeth (Fig. 229). Such spike-like hyperplasias do not occur frequently.

Much more common is the other form of cementum hyperplasia or hypercementosis, which consists of a diffuse thickening of the cementum over a large portion of the root surface (Fig. 230). The clinical significance of this condition has been the subject of a great

deal of consideration and unsubstantiated speculation. Hypercementosis may be found either in all or some teeth of a mouth, especially in those without vital pulps. In generalized hypercementosis, evidently there is a congenital tendency toward abundant deposition of cementum. An observation by Zemsky corroborates



FIG. 229.—Apex of a lower lateral incisor that was subjected to heavy occlusal stress over a long period of time. Spike-like cementum hyperplasias (*CH*) arranged in the direction of the fiber bundles of the periodontal membrane. (Gottlieb, Jour. Am. Dent. Assn.)



FIG. 230.—Cross section through the root end of an intact lower molar with cementum hyperplasia (ground section). Notice the regular arrangement of the successive layers of cementum on the root surface.

this theory. Three members of one family, a mother and two adult daughters, showed radiographically distinct hypertrophy of cementum on the roots of otherwise intact teeth.

It is difficult to give an exact definition of hypercementosis. The cementum is the most variable of all dental structures. In the teeth

of some individuals the cementum is three or four times as thick as it is in those of others; yet this greater thickness has no special significance beyond its being an individual anatomical variation. Therefore, the clinical diagnosis of hypercementosis must be made with caution and with the understanding that it is merely relative: if all of the roots in a jaw appear radiographically thin and slender, except only one or two teeth that have thick, bulky roots, the latter present a condition of relative hypercementosis; but if all of the teeth of one person have thick layers of cementum, the condition could more appropriately be described as well-developed cementum without any special significance. Such a condition is desirable since teeth with thick cementum are usually very firm and are rarely lost by pyorrhea.

Cementum hyperplasia in pulpless teeth has a definite clinical significance: it is a defense reaction of the organism. If a low-grade irritation, originating from within the pulpless tooth, stimulates and irritates the periodontal membrane over a long period of time, the tissues try to wall off this irritation by laying a calcified barricade over the root end. The result is a tooth with cementum thicker than that of the adjacent teeth with vital pulps. The space for the formation of this excess cementum is created by the inflammatory destruction of the alveolar bone. Such a hypercementosis is not in itself a pathological condition; it is merely a reparative and protective reaction and, as such, is useful. If a pulpless tooth had hypertrophy of the cementum, infection of the root canal, but not the cementum hyperplasia, would be the indication for extraction.

The only really practical significance of hypercementosis is the difficulties that may be encountered in extracting such teeth. A root that is slender near the cemento-enamel junction and has a knob-like thickening of cementum near the apex is very likely to fracture during extraction, and it may be necessary to cut away the surrounding alveolar bone to free the apex. But this very difficulty indicates the true biological significance of cementum hyperplasia, namely, a more secure anchorage of the tooth in its socket.

A great number of statements in dental literature refer to possible dangers and consequences of hypercementosis. In reading such data one is led to believe that many of them are simply repetitions of previous statements and have been taken over without due criticism. It is stated that blindness, deafness, paralysis, or insanity may be caused by "hypercementosed" teeth and may be relieved by the removal of such teeth. Up to this time, the author has never seen or heard of any such cure; all that has been found are some general statements that are copied again and again.

The etiology of generalized hypercementosis is unknown. In individual teeth chronic, low-grade periapical inflammation may cause it. Frequently in such teeth there is a marked hypertrophy of cementum adjacent to the area of the apex that is included in the inflammatory process (Fig. 231). The inflammation appears to be the stimulus for cementum formation; the resulting increase of root surface may be considered as compensation for the loss of attachment at the apex. The inflammatory process destroys the

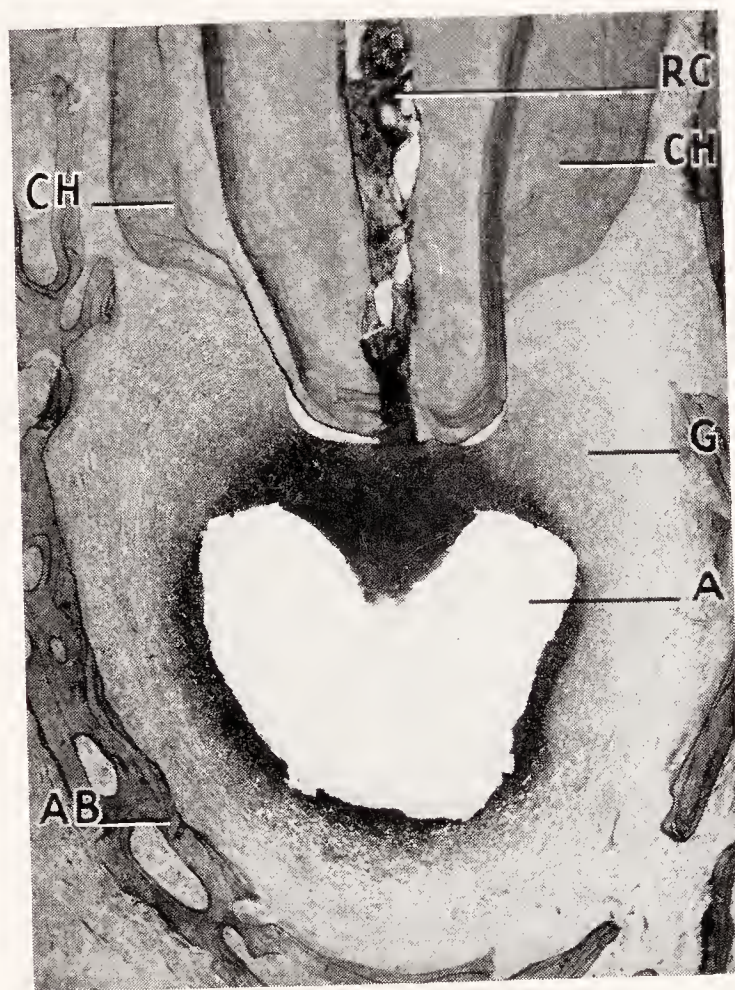


FIG. 231.—Hyperplasia of cementum in the proximity of chronic periapical inflammation. Upper bicuspid. *CH*, cementum hyperplasia; *RC*, root canal containing necrotic pulp remnants; *G*, granuloma; *A*, abscess cavity containing a purulent exudate; *AB*, alveolar bone. (Courtesy of Wm. G. Skillen.)

alveolar bone and the apical fibers and thus weakens the tooth, and the newly built cementum provides a new vital surface for the attachment of additional periodontal fibers.

Functional stimuli do not seem to be of importance in the development of cementum hyperplasias. Increased function does not cause increased thickness of the cementum. Kellner made comparative measurements of the thickness of cementum on corresponding functioning and non-functioning teeth of the same jaw. He found thicker cementum in non-functioning teeth than in those having antagonists. The most striking comparison of this type was found in a completely embedded lower bicuspid and the corresponding lower bicuspid on the other side, which was in normal occlusion: the embedded tooth had thicker cementum than the erupted one. These

observations indicate that function is not of primary importance in hypercementosis.

The changed concept concerning hypercementosis is best expressed in a clinical report by Gardner and Goldstein. These authors reported the following results of their investigation: Of 529 teeth with hypercementosis, 428 were vital and 101 non-vital, or, in other words, 4.2 vital teeth were involved for every non-vital tooth. Gardner and Goldstein drew the following conclusions concerning the etiology of hypercementosis: "This apparently limits the force of any reference to the factor of infection playing a part in the etiology of the hyperplasia, although occasionally we encountered, in examination of non-vital teeth, zones of rarefaction associated with apical hypercementosis. But in each case in which this phenomenon was presented, other vital teeth with hypercementosis were found, pointing to the likelihood of an inherent tendency of certain persons rather than to a hyperplastic response to infection only." In other words, cementum hyperplasia is non-pathogenic and merely indicates an individual tendency toward extensive production of cementum in some teeth. Hence, the authors came to the practical conclusion that "extraction of vital teeth with hypercementosis, in the hope of relieving systemic pathologic conditions, as often practised by dentists, is contraindicated."

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CHAPTER XI.

TOOTH RESORPTION.

GENERAL CONSIDERATIONS CONCERNING HARD TISSUE RESORPTION.¹

NORMALLY, hard tissues are found in the human body in only two structures: bone and teeth. Not all the calcified tissues of these structures, however, have the same origin: bone, dentin, and cementum originate from the mesoderm, enamel from the ectoderm.

The hard tissues are formed in the following manner: an organic matrix is laid down first, and subsequently lime salts are deposited in it. If later such tissues are eliminated, matrix and mineral salts are removed simultaneously. Formerly it was believed that a process of decalcification (halisteresis) preceded the elimination of hard tissues. Koelliker and other investigators, however, have definitely shown that normally the resorptive process in teeth as well as bone is characterized by the presence of special cells, the osteoclasts, that eliminate the calcified tissue without any previous decalcification. True decalcification (halisteresis) occurs only rarely, in severe bone diseases such as osteomalacia.

The origin of the osteoclasts is still a much discussed question. There are two possible sources from which they may develop, either from the capillaries in the connective tissue adjacent to the hard substance, or from the connective tissue itself by the transformation of osteogenetic cells or undifferentiated mesenchyme cells. Typical osteoclasts are large cells (giant cells), containing anywhere from two or three to twenty or thirty nuclei, which are usually clustered in the centers of the large protoplasmic cell bodies (Fig. 232). These osteoclasts lie in bay-like excavations in the hard substance, the Howship's lacunæ.

¹ In modern terminology the term "resorption" for the elimination of hard tissues by cellular activity is given preference over the old term "absorption." As indicated by the prefix, re, resorption means that some structure or substance that is formed or built by the body is taken back into the organism. This is actually true of teeth and bone, which are built by the body and, in case of resorption, are dissolved and taken back into the general circulation. Absorption, on the other hand, means that some new, external material or substance is sucked up or taken into something. It is, therefore, correct to say that moisture is absorbed by cotton or that a drug is absorbed by the intestinal mucosa. (See also Becks and Marshall, Jour. Am. Dent. Assn., 19, 1528, 1932.)

There is apparently no difference between the way in which bone, dentin, cementum, and enamel are resorbed. It is, therefore, unnecessary to call the giant cells associated with tooth resorption odontoclasts or cementoclasts; they are spoken of as osteoclasts.

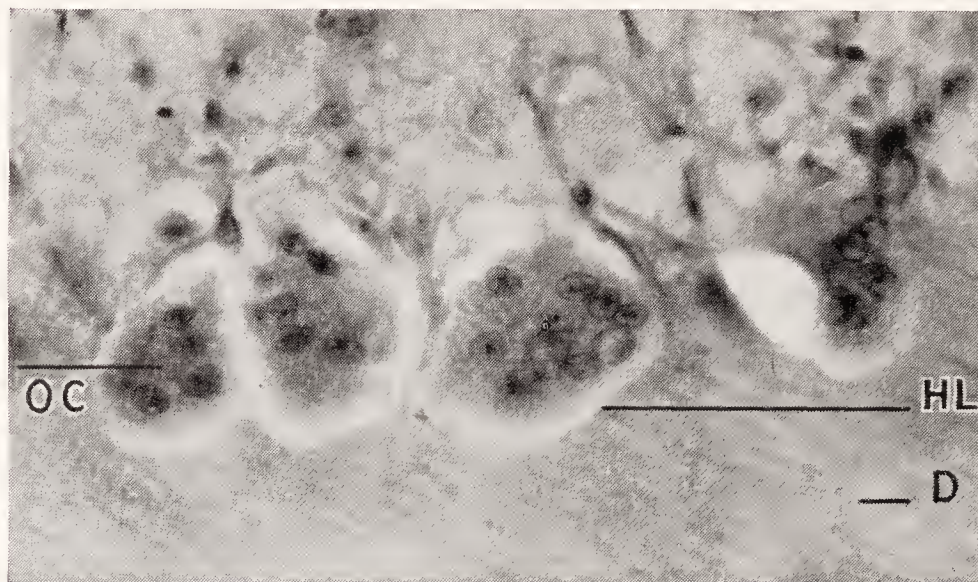


FIG. 232.—Polynuclear osteoclasts (giant cells) resorbing dentin. *D*, dentin; *OC*, osteoclasts with numerous nuclei; *HL*, Howship's lacunæ in the dentin.

PHYSIOLOGICAL RESORPTION ASSOCIATED WITH THE SHEDDING OF THE DECIDUOUS TEETH.

The roots of the deciduous incisors, as a rule, reach their full development at the age of two years, the roots of the cuspids and molars usually about a year later. At about the age of four years, the developing permanent germs lying near and between the roots of the deciduous teeth begin to cause resorption of the latter.

The permanent tooth germ itself has no resorptive properties. It grows and moves and, in so doing, exerts pressure upon the surrounding connective tissue and upon the alveoli and roots of the deciduous teeth. This pressure stimulates resorption of the hard structures lying in the path of the growing permanent tooth germ. The enamel of the tooth germ is protected by the enamel epithelium and therefore cannot be resorbed.

The resorption of the deciduous root usually begins on the side facing the permanent successor. Figure 233 shows the resorption of the root of an upper second molar in a child, aged four and one-half years. The bicuspid germ at this age lies between the roots of the deciduous molar and below the floor of the maxillary sinus. The mesio-buccal root is considerably shortened by resorption; the opening of the root canal is adjacent to the enamel epithelium of the bicuspid crown.

As the permanent tooth grows and moves in an occlusal direction,

the deciduous roots gradually become shorter until finally the crown only is left, superficially attached to the gingivæ. Such a condition is illustrated in Figure 234, in the lower cuspid of a child, aged nine years. A higher magnification shows the lower surface of the crown densely beset with osteoclasts that are resorbing the dentin (Fig. 235). The pulp is intact. The space between the enamel epithelium of the permanent cuspid and the dentin of the deciduous tooth is occupied by a highly vascularized granulation tissue ("absorbent organ" of J. Tomes); after the exfoliation of the deciduous crown, this granulation tissue is found as a bright red, irregular, friable tissue in the area formerly occupied by the deciduous tooth.

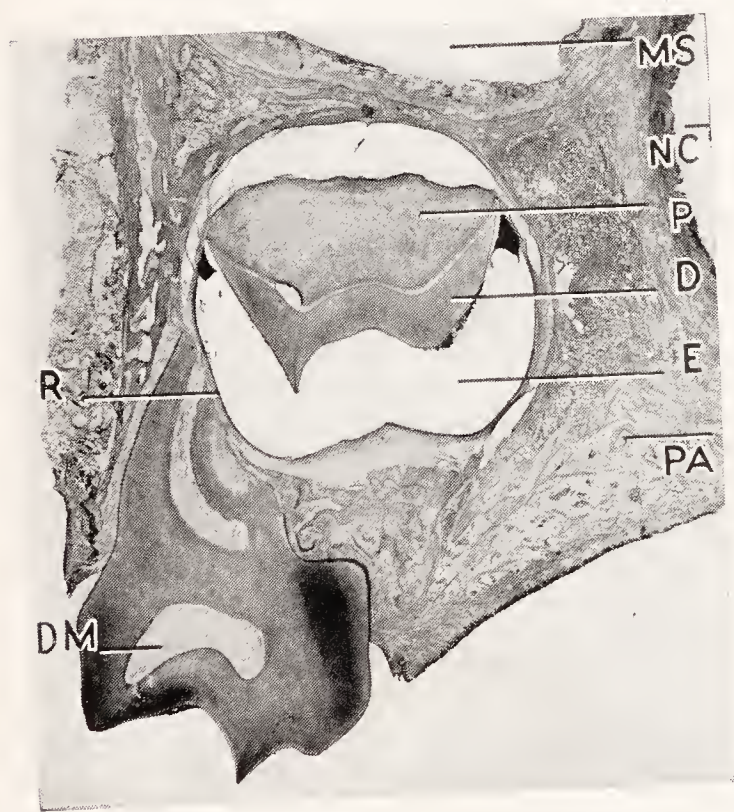


FIG. 233.—Resorption of the mesio-buccal root of an upper deciduous second molar. Bucco-lingual section. Human specimen; age, four and one-half years. *DM*, second deciduous molar; *P*, pulp of the bicuspid germ; *D*, dentin of the bicuspid germ; *E*, enamel of the bicuspid germ; *R*, resorption of the root end of the deciduous molar; *NC*, nasal cavity; *MS*, maxillary sinus; *PA*, palatine artery. (Kronfeld, Dent. Cosmos.)

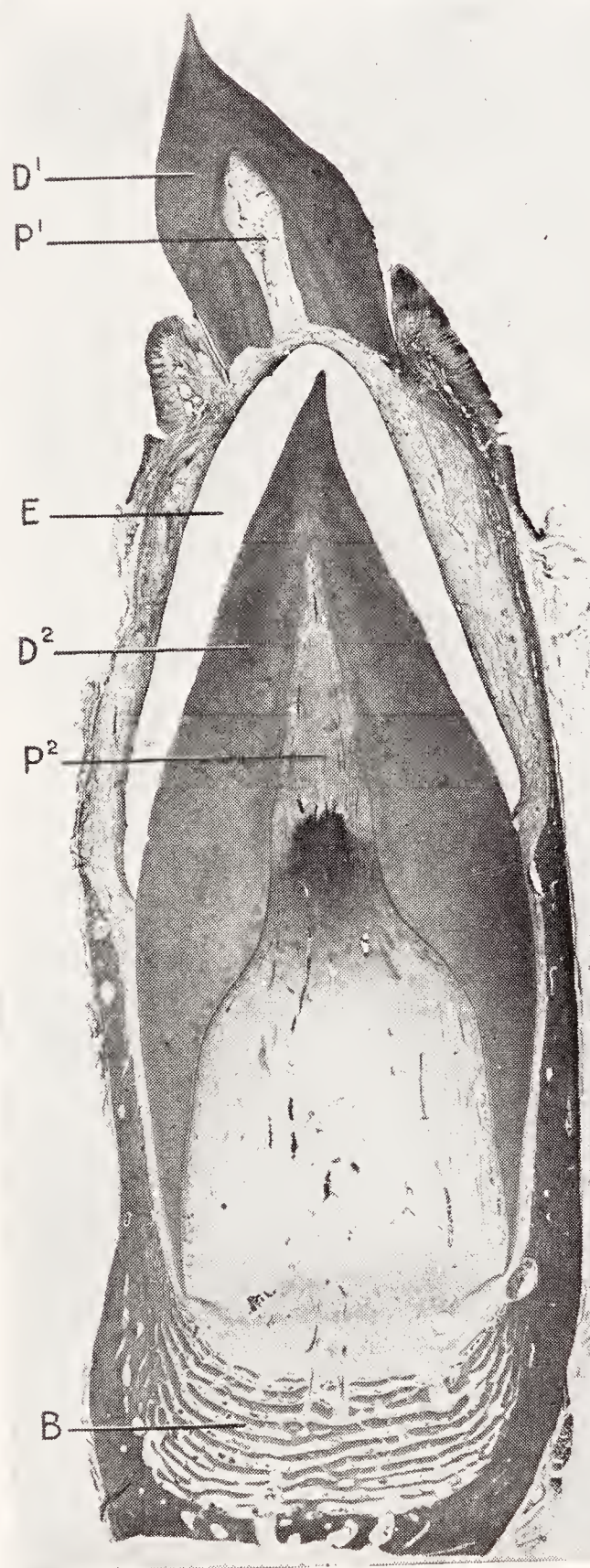


FIG. 234.—Lower deciduous and permanent cuspid. Labio-lingual section. Human, aged nine years. *D*¹, dentin of the deciduous cuspid; *P*¹, pulp of the deciduous cuspid; *E*, enamel of the permanent cuspid; *D*², dentin of the permanent cuspid; *P*², pulp of the permanent cuspid; *B*, rows of bone beneath the apical opening of the permanent cuspid. (Kronfeld, Dent. Cosmos.)

Figure 234 shows an unusual arrangement of the bone trabeculae underneath the permanent cuspid. Each trabecula is being resorbed on the lower surface, whereas new bone is being formed on the upper side, facing the erupting permanent tooth. This distribution of resorption and formation indicates that the permanent cuspid is



FIG. 235.—Higher magnification of Figure 234. *E*, enamel of the permanent cuspid; *D*, dentin of the permanent cuspid; *P*, pulp of the deciduous cuspid; *R*, resorption on the lower surface of the deciduous tooth; *EA*, proliferation of the epithelial attachment along the surface of the deciduous root. (Kronfeld, Dent. Cosmos.)

moving occlusally, away from the fundus of the alveolus; new trabeculae of bone fill the space behind the moving tooth. This corroborates the investigations of Orban concerning the occlusal movement of tooth germs during their development; it shows that the old conception of the root's growing into the underlying bone is incorrect; rather, the entire tooth germ moves occlusally.

The resorption of deciduous teeth does not proceed uniformly from the periphery of the root toward the pulp; the dentin and pre-

dentin immediately surrounding the pulp seem to be more resistant to resorption than the rest of the dentin. Furthermore, the resorptive process itself does not continue with the same intensity throughout the entire period of shedding. The eruptive movement of the permanent teeth is interrupted by periods of inactivity or rest, during which the resorption of the deciduous teeth is also discontinued; then reparative processes take place in the deciduous teeth and alveolar bone. Tomes described this phenomenon as follows: "The process of absorption once commenced does not necessarily proceed without intermission, but may give place for a time to actual deposition of osseous tissue on the very surface eroded. . . . These alternations of absorption and deposition often occur during the normal process of the removal of the deciduous teeth, and result in the deposition of a tissue not unlike cementum in excavations of the dentin, or even of the enamel." Oppenheim, who studied this problem in the teeth of men and animals, found that during the eruptive phase both alveolar bone and deciduous tooth are resorbed to a greater extent than the actual movement of the permanent tooth would necessitate, and that this excess resorption is repaired by formation of new bone and cementum during the period of rest. In the course of this reparative phase, even a solid union between bone and deciduous tooth may develop; this explains the varying firmness of deciduous teeth. During the period of active growth of the permanent teeth, the temporary teeth are rather loose, while during the period of inactivity, they may become firm again.

An extensive bony union between a lower deciduous second molar and the alveolar bone is illustrated in Figure 236. The specimen was obtained from the jaw of a boy, aged eight and one-half years. The fusion between tooth and bone has occurred in the bifurcation of the roots. Clinically, such a deciduous tooth would be very firm, firmer than the adjacent teeth, since the latter would have slight physiological motility. If the bony ankylosis of a deciduous tooth is extensive, it may prevent its normal exfoliation as well as the eruption of the permanent successor.

The occurrence of submerged deciduous molars can also be explained by resorption and subsequent ankylosis with the alveolar bone (Noyes). Such teeth have ceased to move occlusally and remain below the occlusal plane of the other teeth. If they form an obstacle to the eruption of the bicuspid, they should be removed.

In human deciduous teeth, the pulp tissue usually does not participate in the resorptive process; it retains its histological characteristics as pulp and does not become an "absorbent organ." Only in

the last stages of resorption does granulation tissue sometimes proliferate into the deciduous crown and cause resorption on the inside of the pulp chamber. The osteoclasts are found only on the surface of the deciduous tooth that faces the permanent crown (Fig. 235) but not on the inside of the pulp chamber. For this reason deciduous teeth without vital pulps are resorbed and eliminated the same way

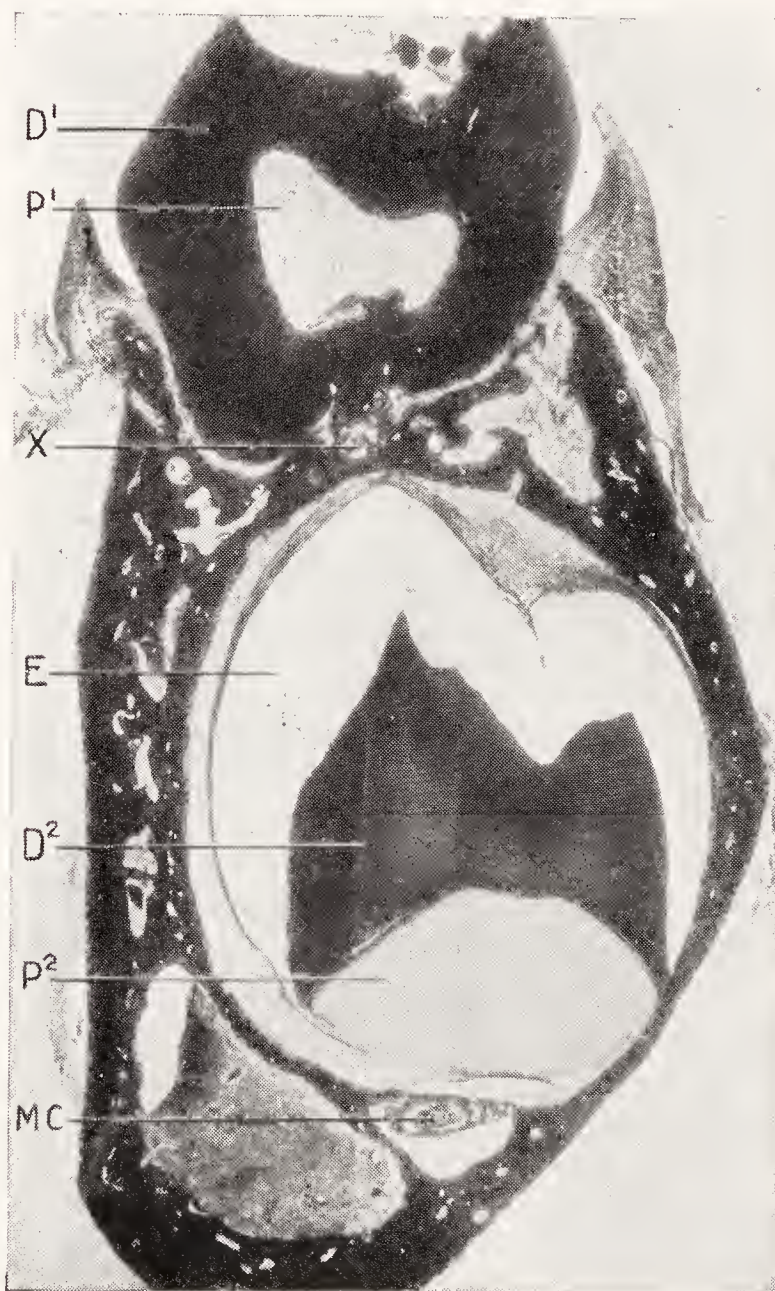


FIG. 236.—Bony union between a lower deciduous second molar and the alveolar bone. Bucco-lingual section. Human, aged eight and one-half years. D^1 , dentin of the deciduous molar; P^1 , pulp of the deciduous molar; E , enamel of the bicuspid germ; D^2 , dentin of the bicuspid germ; P^2 , pulp of the bicuspid germ; X , area of bony union; MC , mandibular canal. (Kronfeld, Dent. Cosmos.)

as deciduous teeth with intact pulps, provided no infection is present.

The resorption of deciduous teeth with infected root canals is much slower and more irregular than that of teeth with vital healthy pulps. Infected, necrotic hard tissues are very resistant to resorption; instead they are eliminated *in toto*, like a sequestrum.

Infected deciduous roots are pushed out by the permanent successors instead of being gradually resorbed.

Resorption of Deciduous Teeth Without Permanent Successors.—

Primarily the resorption of the roots of deciduous teeth is controlled by and dependent upon the permanent successors. But this mechanical explanation is insufficient to explain the resorption of the roots of deciduous teeth without permanent successors. In the latter root resorption takes place without the stimulus of the permanent tooth; therefore, obviously the presence of the permanent successor is not the only cause of resorption of a deciduous tooth (Fig. 16).

Clinical observations show that the histories of retained deciduous

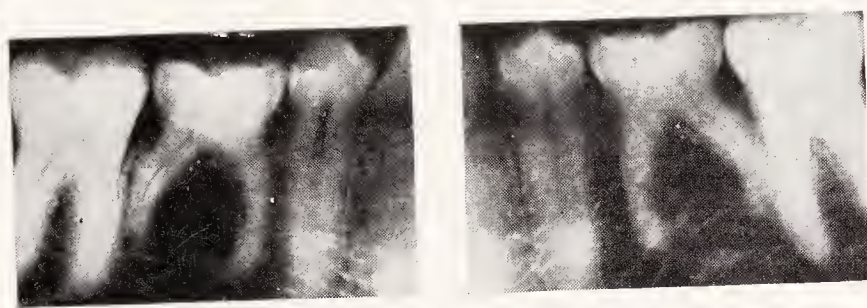


FIG. 237.—Radiographs of a patient, aged forty years, with retained lower deciduous molars. No second bicuspid are present. Notice the areas of resorption on the mesial roots of the deciduous molars. (Courtesy of G. E. Morgan.)

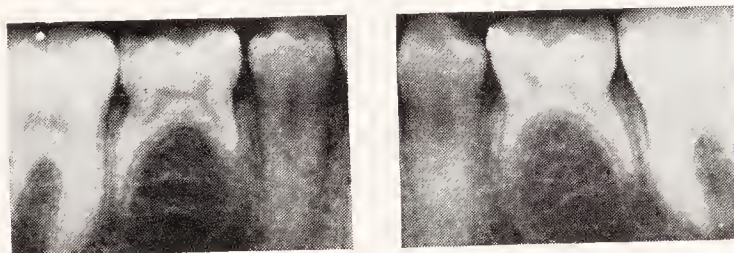


FIG. 238.—Radiographs of a child, aged thirteen years, with retained lower deciduous molars. No second bicuspid are present. The roots appear to be considerably shortened by resorption. (Courtesy of G. E. Morgan.)

teeth vary widely. If the permanent successors are embedded or missing, the deciduous teeth, especially cuspids and molars, are often retained for years in good condition. Sometimes such deciduous teeth become loose and are lost for no apparent reason; their roots are extensively resorbed, exactly like those of normally shed deciduous teeth. It seems that the condition of the deciduous tooth itself decides the final outcome. A root is retained in its socket only as long as it has a vital cementum surface. As long as the deciduous tooth has such a surface, a good functional connection with the alveolus is assured. Small resorptions are repaired by new deposits of cementum. How long this favorable condition will last cannot be predicted (Figs. 237 and 238). In many cases of this type the vitality of the deciduous tooth seems to be lost in the second or third decade of life. No deposits of new cementum take place on the root surface,

and resorptions are no longer repaired; instead the resorptive process continues until the deciduous tooth is loosened and finally lost. These changes in the tooth's vitality are an expression of senescence, and as such are largely independent of outer influences.

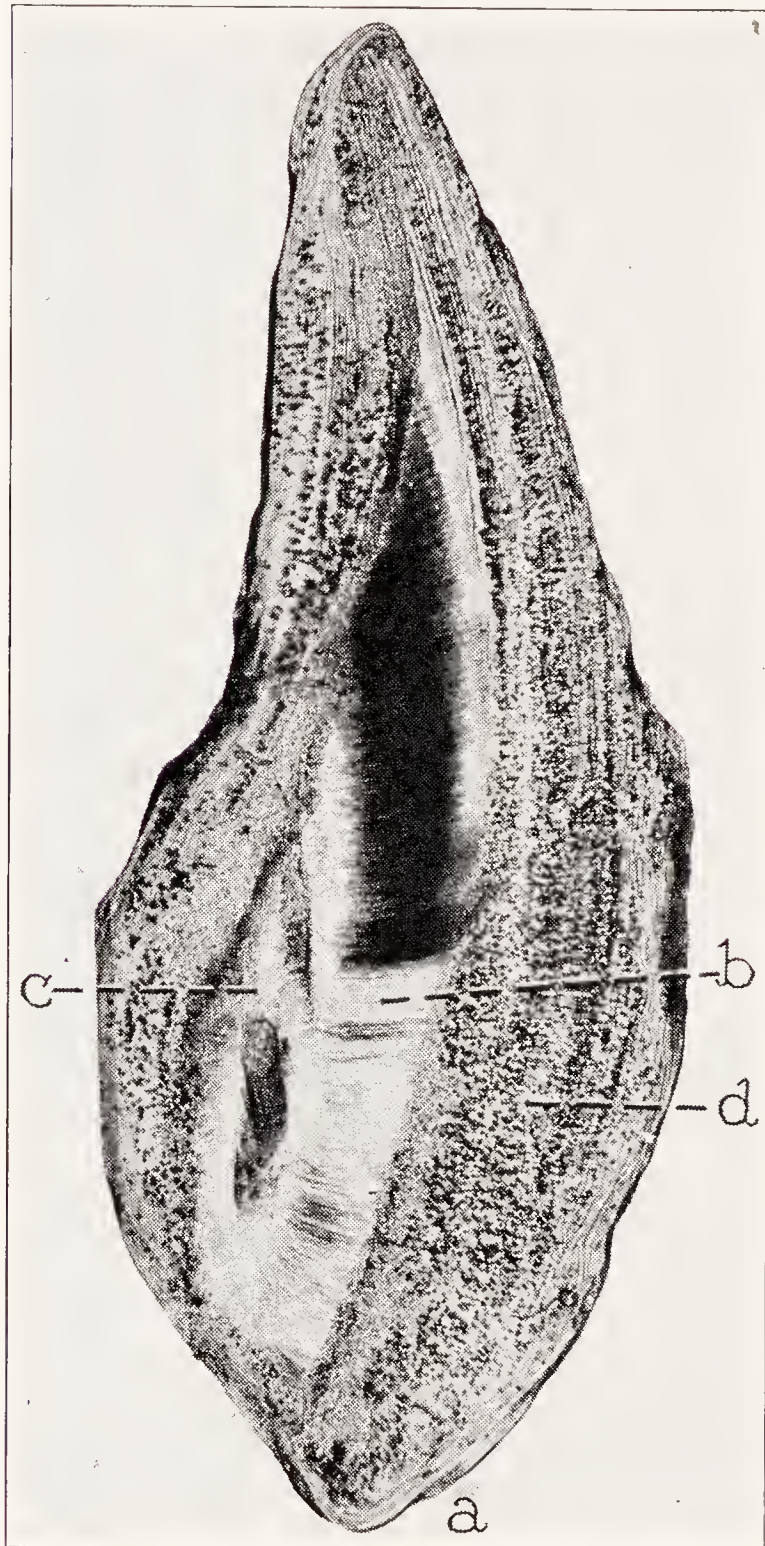


FIG. 239.—Ground section through a retained deciduous root tip from an adult patient. *a*, apical end; *b*, dentin; *c*, root canal; *d*, hyperplastic cementum. (Austin and Stafne, Jour. Am. Dent. Assn.)

Retained Deciduous Root Ends.—In the radiographic and histological examination of the jaws of adult persons, small radiopaque bodies are occasionally found in the alveolar bone between the roots of the permanent teeth. Most commonly they are found in the area of the second bicuspid. Kotanyi and Schönbauer have studied

such structures *in situ* in sections through human jaws; they recognized them as root fragments of deciduous teeth that had remained embedded in the jaw, either surrounded by periodontal membrane or fused to the bone. Austin and Stafne removed several such root tips surgically. They were covered by thick layers of cell-containing cementum (Fig. 239).

The presence of such retained root tips of deciduous molars can be explained by the mechanism of their resorption. The erupting bicuspid advances toward the bifurcation of the deciduous molar and resorbs the under surface of the roots. In doing so, one or two apices of the deciduous molar may be cut off by resorption; they become separated from the rest of the root and may be left behind in their original position in the jaw, while the crown is exfoliated.

RESORPTIVE PROCESSES OCCURRING IN PERMANENT TEETH.

Resorptive processes in permanent teeth are always the expression of some pathological process. The several causes of such resorptions will be enumerated, and each group will be discussed both from the clinical and the histopathological viewpoint. The kinds of teeth occasionally subject to resorption are:

Pulpless teeth.

Replanted teeth.

Embedded teeth.

Teeth in close proximity to tumors and cysts of the jaws.

Teeth subjected to excessive occlusal trauma.

Teeth with resorption of unknown etiology (Idiopathic resorption).

Root Resorption in Pulpless Teeth.—A rather frequent clinical observation, usually revealed by the radiograph, is that roots may be resorbed following death and infection of the pulp. The resorption begins either at the root end and involves the apical part of the root, or on the side of the root and progresses toward the root canal.

A large percentage of pulpless teeth with chronic periapical infection shows some resorption at the apex. If the root canal is successfully filled, these areas of resorption are covered by a reparative deposit of new cementum.

A case of extensive root resorption was observed by Willman in a boy, aged fourteen years, whose upper left central incisor gradually became shorter than the neighboring teeth (Fig. 240). The radiograph revealed extensive destruction of the root; there was little left besides the guttapercha root canal filling (Fig. 241). The

microscopic examination of the tooth after surgical removal showed the amount of resorption of the root and the subsequent ingrowth of alveolar bone (Fig. 242). By the growth of bone into the resorbed area the tooth had become anchored to its socket, and its further eruption had been prevented; the neighboring teeth continued their occlusal movement, which resulted in the apparent shortening of the left central incisor.

Root Resorption in Replanted Teeth.—Extracting and replanting a tooth almost invariably cause root resorption. Regardless of whether or



FIG. 240.—Plaster cast of the upper anterior teeth of a boy, aged fourteen years, showing the upper left central incisor approximately 2 mm. shorter than the neighboring teeth.



FIG. 241.—Radiograph of the upper central incisor. The pulp had been removed and the canal filled two years before. Most of the root has been destroyed by resorption.

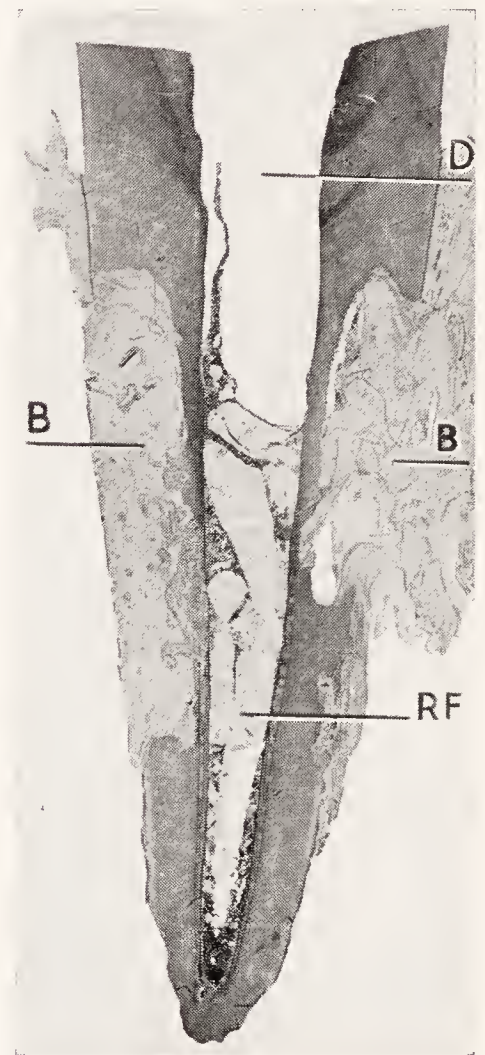


FIG. 242. — Mesio-distal section through the root of the upper left central incisor showing large areas of resorption with ingrowth of bone. *B*, bone; *D*, space formerly occupied by the dowel supporting the porcelain crown; *RF*, root canal filling. (Willman, Jour. Am. Dent. Assn.)

not the root canal of the replanted tooth has been filled, a radiographic examination after a period of several years always reveals more or less extensive resorption. Sometimes ingrowing bone replaces the resorbed dentin and holds the replanted tooth firmly in place; then it may give good service for a long time. In other cases, no such repair takes place; the replanted tooth becomes loose, and when it is finally eliminated there is practically no root left.

A dog's incisor was extracted, the pulp removed, and the canal filled with guttapercha. The tooth was then replanted and held in place by wires for three months. Microscopic examination shows the beginning of resorption of the root (Fig. 243). In some places root surface and alveolar bone have become fused, providing good anchorage of the tooth in the jaw, at least temporarily (Fig. 244).

The high incidence of root resorption in replanted teeth suggests that the extraction and manipulation of the tooth may damage the



FIG. 243



FIG. 244

FIG. 243.—Replanted lower incisor of dog. The tooth was extracted, the root canal filled with guttapercha, and the tooth replanted and held in place by wires. Note the extensive resorption of the root at *R*. At *B*, root and bone are grown together. *G*, guttapercha cone

FIG. 244.—Higher magnification of the bony union. From *A* to *B* the original cementum surface is united with the bone; from *B* to *C* the union has taken place following root resorption.

(From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

root surface to such an extent that it becomes subject to resorptive and reparative changes like those that occur in a piece of ivory or bone that has been implanted in the jaw.

Resorptive Processes Associated With Embedded Teeth.—The pathology of embedded teeth will be discussed in a later chapter of this book; here only the possibilities of tooth resorption in connection with embedded teeth will be considered. Two forms of resorption associated with embedded teeth must be distinguished, namely,

resorption of the embedded tooth itself, and resorption of neighboring teeth which is caused by the embedded tooth.

Resorptive Processes Occurring in Embedded Teeth.—The examination of embedded teeth occasionally reveals resorptive processes that have begun either on the crown or on the root (Fig. 245). However, the resorption of completely embedded teeth is not a frequent occurrence. Much more often they are found to be intact, having a smooth, normal surface, even though they have been embedded for decades. Normally the enamel epithelium of the

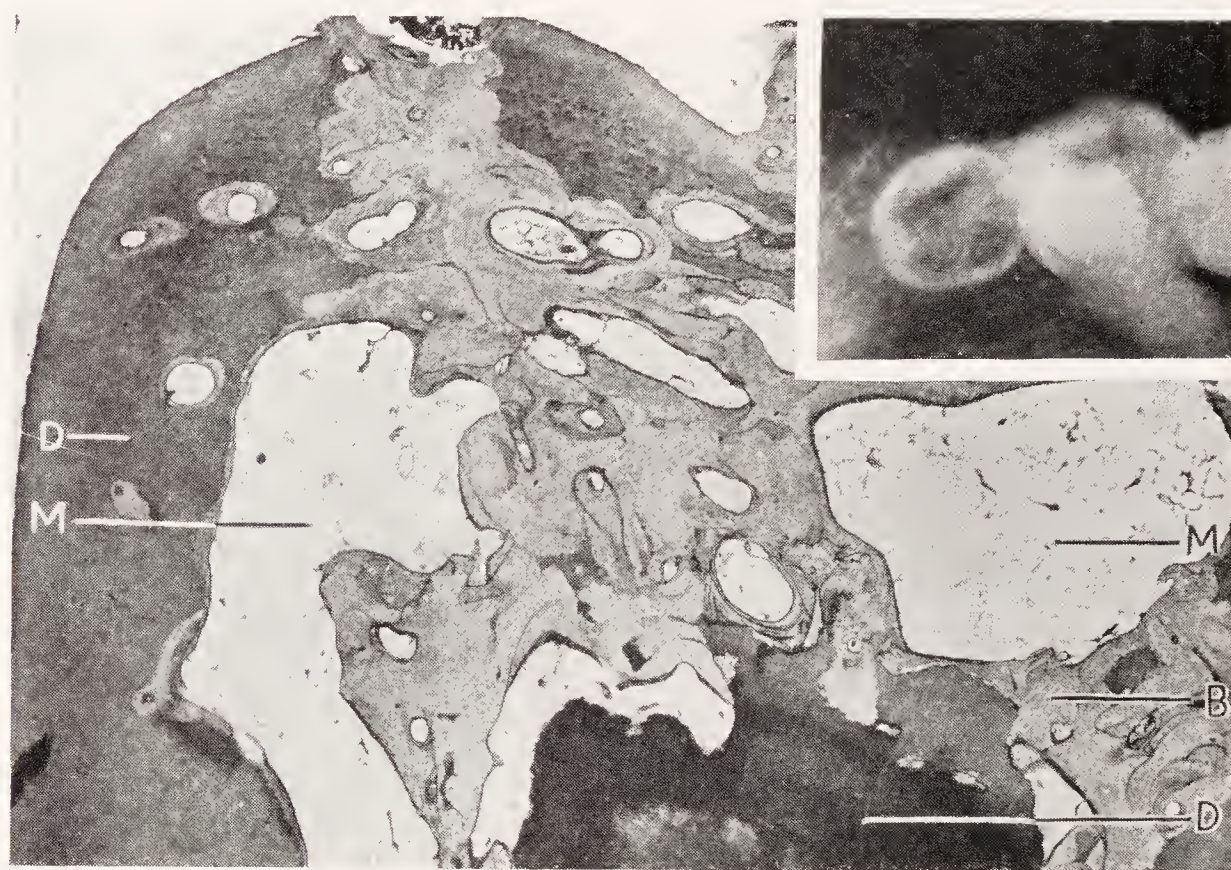


FIG. 245.—Resorption of a completely embedded lower molar. The radiograph in the upper right corner shows extensive defects in the crown of the tooth. The specimen shows replacement of most of the dentin by bone and bone marrow. *D*, dentin; *B*, bone; *M*, fat marrow. (Courtesy of W. H. G. Logan.)

unerupted tooth protects the enamel by separating it from surrounding connective tissue. In order to make it possible for the crown of an embedded tooth to be resorbed, the enamel epithelium covering the crown must be destroyed, exposing the enamel to the surrounding connective tissue. Inflammation spreading from the infected root canal of a neighboring erupted tooth may cause destruction of the enamel epithelium.

Worman studied the jaws of a dog containing several unerupted, embedded teeth and found evidence of resorption in all of them. Figure 246 shows an embedded, malformed premolar of this dog. The crown is small, and the root broad and short. Alveolar bone and enamel have become united; the dentin shows several large areas

of resorption extending close to the pulp. A higher magnification of the crown surface shows the deposition of bone upon the irregularly resorbed enamel surface (Fig. 247). Several findings in these speci-



FIG. 246.—Embedded lower premolar of dog showing extensive resorption. *E*, enamel; *B*, bone deposited upon the enamel; *D*, dentin; *R*, resorption of dentin; *PC*, pulp chamber. (Worman, Jour. Am. Dent. Assn.)

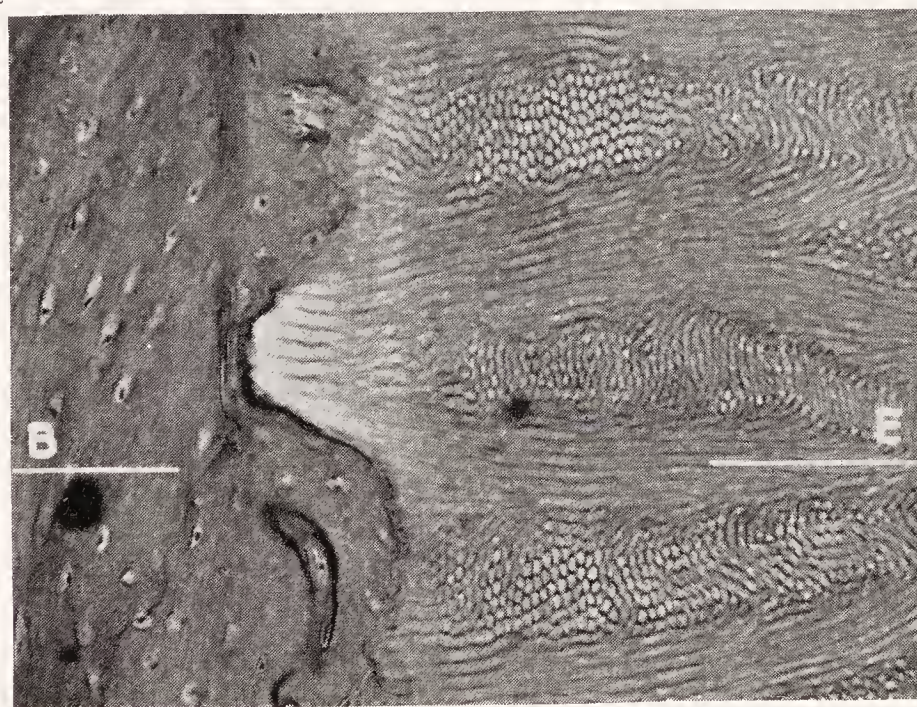


FIG. 247.—Higher magnification of Figure 246. Deposition of bone, *B*, on the irregularly resorbed surface of the enamel, *E*. (Worman, Jour. Am. Dent. Assn.)

mens support the opinion that the atrophy of the protecting enamel epithelium occurs first; then, when the enamel is exposed to the periodontal connective tissues, resorption takes place.

From time to time in dental literature appear reports of what the observer calls "caries" in embedded teeth. These reports are based on a failure to distinguish between caries and resorption. Caries cannot occur in a completely embedded tooth. In the radiograph resorptive defects in the crown of an embedded tooth may look like caries, but the microscopic examination of the extracted specimen invariably reveals the presence of osteoclasts and Howship's lacunæ in the enamel and dentin and thus establishes the diagnosis of resorption.

Resorption of the Roots of Neighboring Teeth Caused by Embedded Teeth.—In their attempt to erupt, embedded teeth often move with considerable force in the direction of the roots of adjacent erupted teeth, causing resorption of the roots of the latter. The most common occurrences of this type are resorption of the root of the lateral incisor by the crown of an embedded cuspid, and the resorption of the distal root of the lower second molar by the crown of an embedded lower third molar. Clinically, the progress of such resorptions is often indicated by soreness, loosening, or displacement of the erupted tooth on which the embedded tooth impinges. The movement of an embedded tooth is not continuous but interrupted by periods of inactivity and rest; periods in which there are clinical manifestations of pressure caused by the embedded tooth may be followed by symptomless periods. During the rest periods the resorbed area may be repaired by newly deposited cementum. Such a reparative deposit of cementum also takes place if the embedded tooth is removed surgically.

Tooth Resorption in the Presence of Tumors and Cysts of the Jaws.—Any pathological growth in the jaws may cause resorption of the teeth in its vicinity. The extent and rate of resorption depend largely upon the type of growth. A benign growth (dentigerous cyst, dental root cyst, epulis) is more likely to displace teeth than to cause root resorption. A malignant tumor, because of its rapid and destructive growth, may produce rapid and extensive resorption of the roots that lie in the direction of its expansion.

The mechanism of tooth resorption in such cases is the same as in the resorption caused by an embedded tooth. The tumor causes pressure upon the connective tissue surrounding the root, stimulating this tissue to osteoclastic activity. This is particularly true of tumors of epithelial origin (ameloblastoma, carcinoma). Their epithelial tissue has no resorptive properties; it merely stimulates, by its growth, the resorptive activity of the connective tissue between tumor and root (Euler, Hofer).

In all cases of resorption of teeth by growths in the jaws, resorptive and reparative processes alternate or are found simultaneously in different parts of the same tooth. It seems that either the growth of the tumor is not continuous, making possible temporary reparative action, or a change in the direction or rate of tumor growth temporarily relieves the pressure.

The resorption of a lower cuspid by a rapidly growing malignant tumor of mesodermal origin (osteogenic sarcoma) is illustrated in Figure 248. During a period of less than a year, the patient, a young woman, developed a large growth that involved the entire anterior portion of the mandible. When the lower right cuspid was removed in biopsy the pulp was found to be vital, although more than one-half of the root had been destroyed.

If the destruction involves only a small portion of the root, it is sometimes possible to retain a tooth after the growth has been removed surgically. Figure 249 shows a lower incisor located in an area formerly occupied by a cystic ameloblastoma. The tumor had been removed surgically several years before. The resorbed lower surface of the root is covered

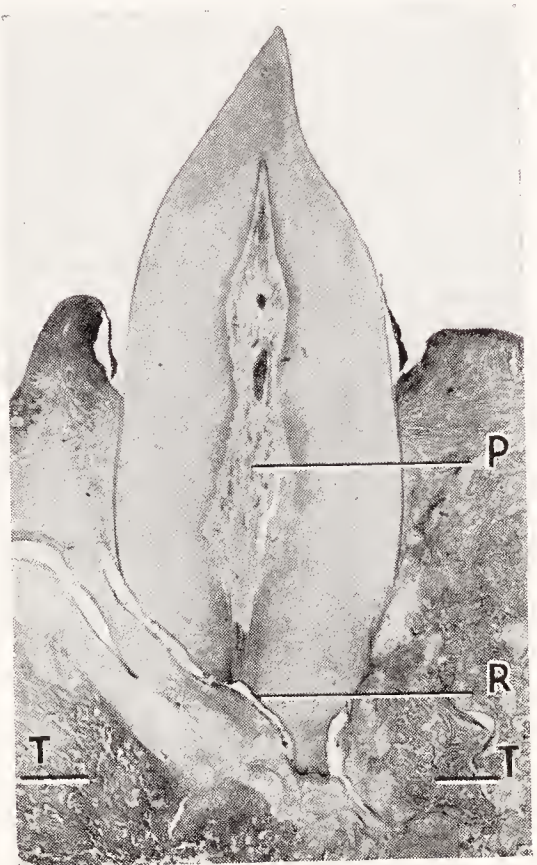


FIG. 248.—Resorption of the root of a lower cuspid by a malignant tumor (osteogenic sarcoma). *P*, pulp of cuspid; *R*, resorbed surface of the root; *T*, tumor consisting of large irregular masses of bone and cartilage.

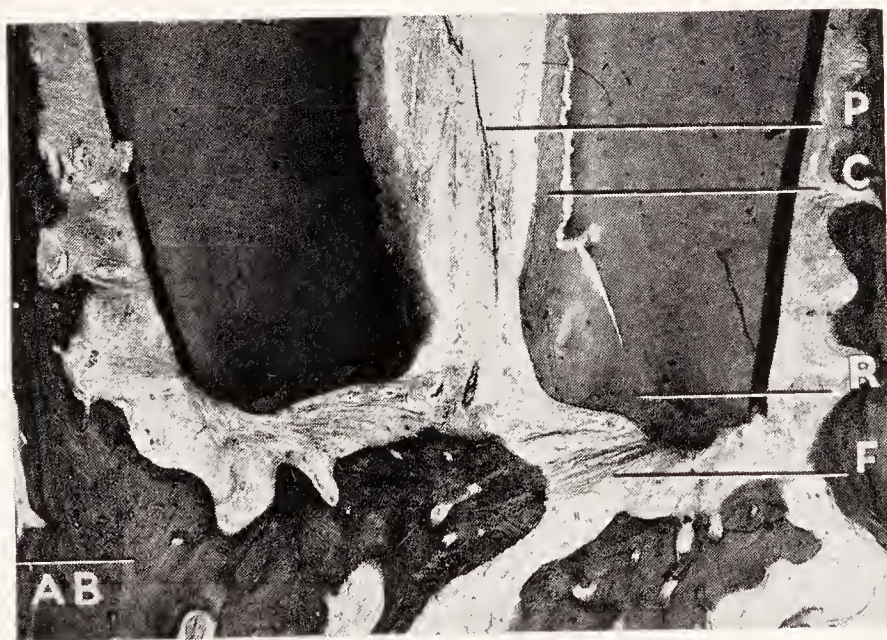


FIG. 249.—Repaired resorption of the root of a lower incisor. A cystic tumor had been removed from this area several years before. *P*, pulp; *C*, deposits of cementum on the walls of the root canal; *R*, resorbed root surface covered by newly formed cementum; *AB*, alveolar bone; *F*, fiber bundles running from the newly deposited cementum to the alveolar bone. Through these fiber bundles a functional connection between root and bone has been reestablished.

with reparative deposits of cementum; the periodontal membrane has been regenerated around all of the root; strong fiber bundles run from the newly formed apex to the fundus of the alveolus. Cementum has been deposited in the apical portion of the pulp chamber, and the pulp tissue is vital.

Root Resorption in Teeth Subjected to Excessive Occlusal Trauma.—The experiments of Gottlieb and Orban on the influence of excessive stress upon the teeth of dogs have shown that occlusal

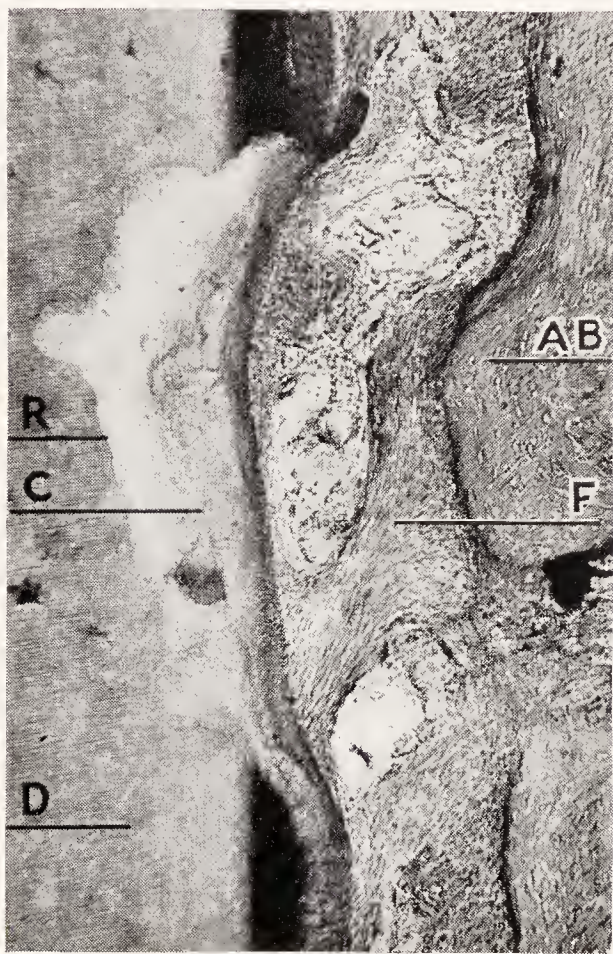


FIG. 250.—Repaired resorption on the root of an upper molar. Alveolar bone and periodontal membrane have been completely regenerated in the resorbed area. *D*, dentin; *R*, line of resorption in the dentin; *C*, reparative deposits of cementum; *F*, fiber bundles of the periodontal membrane; *AB*, alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

trauma of long standing is able to cause root resorption. Similar resorptions have been found in human jaws under identical conditions. Evidence of resorption on the surface of the root is frequently found in areas of pressure. Most of these resorptions are repaired by newly formed cementum, so that they should be considered scars rather than actual injuries. It must be assumed that at some time an occlusal trauma had occurred in such teeth that caused resorptions, which were subsequently repaired (Fig. 250). Sometimes a minute trauma, such as that caused by biting upon a hard object, may be responsible for slight damage to the periodontal membrane and subsequent root resorption in the damaged area. Such minor traumatic injuries are probably responsible for most small repaired root resorptions.

Whenever resorption of the root takes place the periodontal fibers lose their attachment to the involved area of the root surface; the corresponding alveolar bone is also resorbed, and the periodontal membrane becomes wider in the damaged area. When repair takes place, new cementum with new fibers is deposited on the resorbed root surface; functional stimuli are again transmitted to the bone, which is regenerated until the original thickness of the periodontal membrane is restored. This is called functional repair. Often the

reparative formation does not restore all of the original root surface, in which case the bone projects into the depressed area of the root, making the width of the periodontal space uniform.

Orthodontic tooth movement is invariably accompanied by small, circumscribed root resorptions. These resorptions are repaired as soon as active movement is discontinued (Chapter XVI).

Tooth Resorption of Unknown Etiology (Idiopathic Resorption).—

From time to time root resorptions of unknown etiology are observed in permanent teeth with intact pulps. The term "idiopathic root resorption" has been applied to this condition. Sometimes only one tooth is resorbed; sometimes several teeth are involved. Occasionally the case history reveals some accident (trauma) that might have started the process; a small traumatic resorption, instead of being repaired, may have become larger and gradually involved the entire root. Usually, however, the etiology is unknown. Only careful observation and close coöperation with investigators in the field of internal medicine may eventually lead to a solution of this problem.

Idiopathic Resorption of Individual Permanent Teeth.—A case of extensive resorption was observed in the upper first bicuspid of a boy, aged fifteen years. The tooth had a pink color. The radiograph disclosed a very indistinct root outline and an abnormal configuration of the pulp chamber. The tooth was in normal occlusion, the crown intact; all other teeth in the mouth were normal. The bicuspid was removed. In a radiograph of the specimen, the dentin of both the root and the crown appears to have been replaced by cancellous bone (Fig. 251), a fact confirmed by the microscopic examination. Almost all of the dentin was replaced by fibrous connective tissue and newly formed bone trabeculæ. In some places the process of dentin resorption was still going on. Of the crown only a thin outer cap of enamel was left, which also had been attacked by the resorptive process. The pink discoloration of the tooth was caused by a highly vascular soft tissue inside the crown that shone through the thin, transparent enamel ("pink spots" of Mummery). No cause was found for the resorption.

Another case of tooth resorption of unknown origin is illustrated in Figure 252. Clinically, two upper central incisors of a man, about fifty years of age, were loose, although the other teeth were in fairly good condition. The radiograph revealed that little except the crowns was left of the central incisors, which were attached only by the gingiva. The area normally occupied by the roots had been filled in by bone. The patient could give no information as to a

possible cause for this condition; he asserted that until a short time before the teeth had been firm and normal in every respect. The histological examination showed normal crowns and normal pulp chambers. The roots ended abruptly in the gingival third; the root

ends had been covered by a layer of cementum to which periodontal fibers were still attached. It seems that after most of the roots had been resorbed a repa-

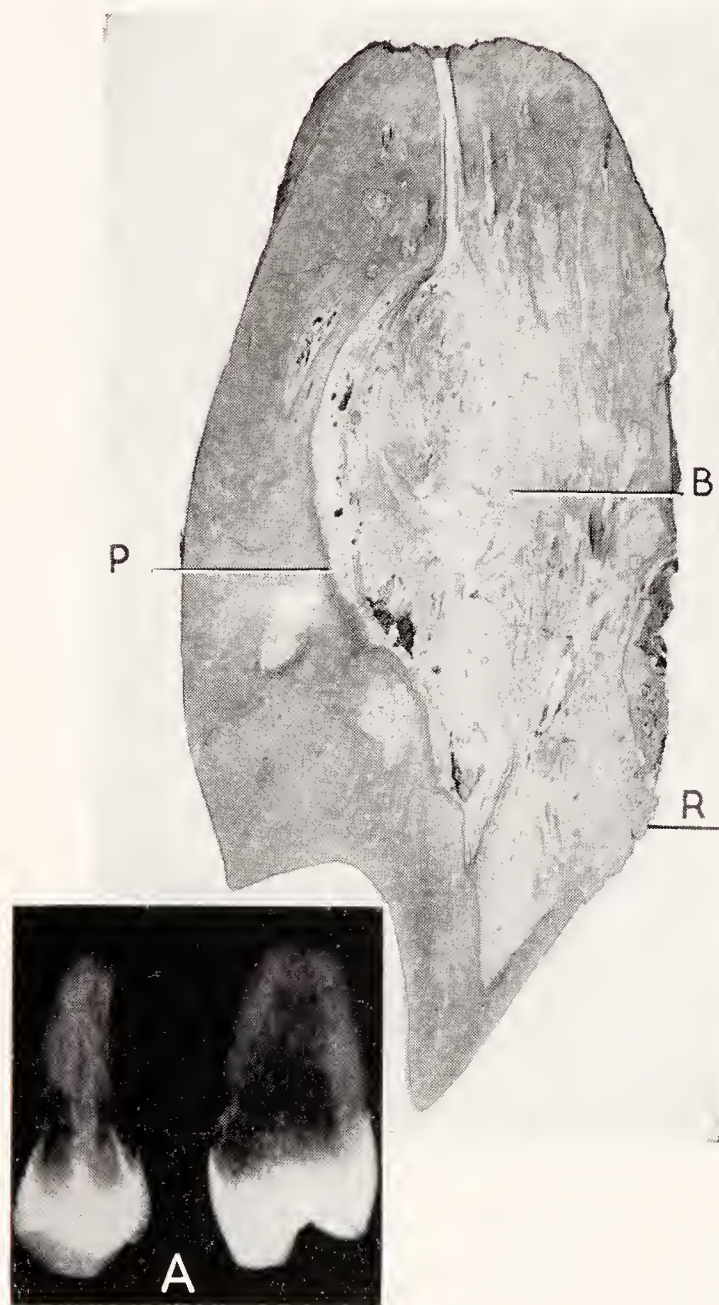


FIG. 251.—Extensive resorption of an upper first bicuspid of a boy, aged fifteen years. Formation of bone in the resorbed area. Etiology unknown. *A*, radiographs of the extracted tooth. Bucco-lingual section through the specimen. *P*, original outline of pulp chamber; *B*, connective tissue and newly formed trabeculae of bone replacing the pulp; *R*, resorption of enamel. (Courtesy of D. M. Gallie, Jr.)

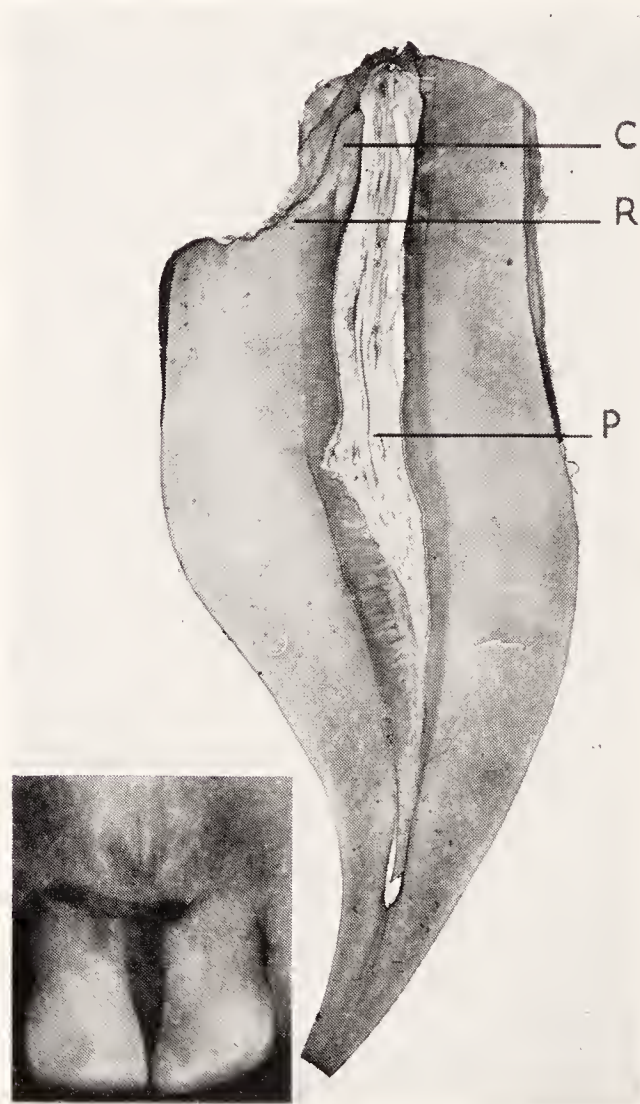


FIG. 252.—Resorption of the roots of the upper central incisors of a man, aged fifty years. Radiographically the roots appear very short. Clinically the teeth were moderately loose. *P*, pulp; *R*, resorption on the root; *C*, cementum covering the resorbed root surface.

rative deposit of cementum occurred and a new attachment was formed, but that the small area of attachment was not large enough to retain the crowns, and the teeth were lost.

A special form of idiopathic root resorption is known as chronic perforating hyperplasia of the pulp, internal resorption or internal

granuloma. Essentially it is the presence of granulation tissue within a tooth, apparently with resorption of the dentin from the inside outward. In a radiograph a sharply outlined defect is found inside of the tooth, which may or may not appear to connect with the shadow of the periodontal space (Figs. 253 and 254).

As a rule, the clinical diagnosis of internal resorption is not difficult. Any radiographically visible defect in a tooth that is not caused by caries must be the result of a resorptive process of some kind. More difficult is the pathological interpretation. Two possibilities exist: Either the resorption originated from the pulp, began inside of the tooth, and progressed from the pulp cavity outward, or it originated in the periodontal membrane and invaded the pulp chamber from without. In advanced destruction of the tooth the origin can no longer be ascertained, since the granulation tissue in the tooth communicates with similar tissue in the periodontal membrane, and



FIG. 253



FIG. 254

FIG. 253.—Upper left lateral incisor with a mesio-distal defect on the root. (Applebaum, *Dent. Cosmos*.)

FIG. 254.—Bucco-lingual view of lateral incisor shown in Figure 253, after extraction. (Applebaum, *Dent. Cosmos*.)

it is impossible to decide where this tissue originated. But there are early cases on record in which the condition was actually confined to the inside of the tooth (Vandory). A chronic inflammatory process was present in the pulp tissue, transforming it into granulation tissue and resorbing the walls of the pulp cavity. In the majority of cases reported in the literature, the resorption probably began on the outside of the root, near the cemento-enamel junction, and from there progressed inward toward the pulp (Applebaum, Göllner). Sometimes reparative changes take place, and newly formed cementum and bone may be found in the cavity produced by the resorption.

No treatment is known for this kind of idiopathic resorption. Sometimes the resorptive process stops spontaneously; then the

pulp retains its vitality and the tooth can be saved. More often, however, the destruction gradually becomes so extensive that the tooth is lost.

Generalized Idiopathic Root Resorption.—Several cases of generalized root resorption have been studied and described histologically. In some there is at least a probability that systemic disturbances were responsible for the destruction of the teeth. One patient, a woman thirty-eight years of age, was observed over a period of two years, during which time extensive resorptions developed on the roots of several intact permanent teeth (Mueller, Rony, and Kronfeld). The radiographs of some of these teeth may give an idea of the rapidity of the destructive process (Fig. 255). In January, 1929, a shallow defect was visible near the alveolar crest on the mesial side of an



FIG. 255.—Extensive resorption of root and crown of the upper right second bicuspid of a woman, aged thirty-eight years. The three radiographs in the right lower corner show the rapid progress of destruction. *A*, January 19, 1929; shallow indentation on the distal side of the crown. *B*, March 6, 1929; indentation on both the mesial and distal side of the root. *C*, May 27, 1929; in less than three months the defects visible in *B* have met in the middle of the tooth, so that crown and root appear to be almost entirely separated. Mesio-distal section through the crown portion of the bicuspid: *D*, dentin; *E*, enamel; *G*, gingival tissue; *R*, resorption undermining the enamel; *GT*, granulation tissue; *P*, pulp; *D'*, predentin and dentin surrounding the pulp. (Mueller, Jour. Am. Dent. Assn.)

upper second bicuspid. On March 6, a similar defect had appeared on the distal side of this tooth. About three months later, on May 27, these two defects extended through the entire thickness of the root, thus separating root and crown. Figure 255 shows a mesio-distal section through the crown. The resorptive process had undermined

the enamel of the crown. A higher magnification of the inside of the crown illustrates the large number of giant cells, which explains the unusual rapidity with which the dentin of this tooth was being resorbed (Fig. 256).

The lower right cuspid and first bicuspid were observed radiographically over a period of about seven months. They showed the same rapid process of root destruction between January and July, 1929. A specimen consisting of these two teeth was removed in July, 1929. It showed evidence of the beginning of repair; deposits of a bone-like substance (cementum) were found inside the hollow crown. In some areas where the undermining resorption of the

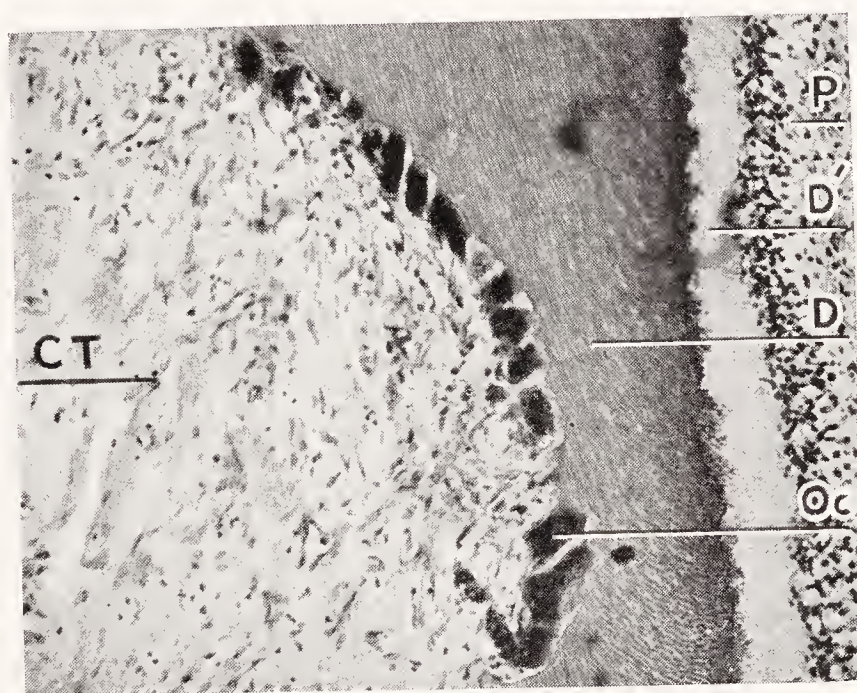


FIG. 256.—Higher magnification of the dentin resorption in Figure 255. The entire dentin surface is densely beset with polynuclear osteoclasts (giant cells). *P*, pulp; *D'*, predentin; *D*, dentin; *Oc*, osteoclasts lying in Howship's lacunæ in the dentin; *CT*, connective tissue. (Mueller, Jour. Am. Dent. Assn.)

crown had already attacked the enamel from the inside, extensive deposits of cementum had occurred upon the inner enamel surface.

In the case described above a disturbance in the liver function was found. In the summer of 1929 treatment of the liver disorder was started. From that time on the resorptive process in the teeth came to a standstill; no more teeth were attacked, and the destroyed areas were reduced by deposits of hard tissue. Whether the dental condition and the hepatic disorder were related etiologically remains, of course, to be proved; such proof could be established only by the observation of additional cases of this kind.

In the case of generalized root resorption reported by Gottlieb in 1923, the patient, a man, aged twenty-two years, had died of influenza. Except for the upper first bicuspid and the lower first molars, all crowns were intact. A microscopic examination of the

teeth showed superficial resorptions on all of the roots; in some places reparative processes had begun (p. 348).

Recently Soifer reported extensive resorption and replacement of the pulp and dentin by fibrous tissues in several anterior teeth of the same individual. The etiology of the resorption was unknown. The root resorptions observed by Becks in hypothyroid patients also belong in the group of resorptions of systemic origin. In some of these patients, thyroid medication was followed by a cessation of the resorptive process.

The knowledge about idiopathic root resorption is still very limited. Only a few cases have been studied, and each has offered a different clinical and microscopic picture.



FIG. 257.—Ankylosis (bony union) between root and bone from chronic periapical inflammation. *G*, granuloma at apex; *R*, resorption of dentin; *A*, bridge of bone extending from alveolar process to dentin; *B*, alveolar bone. (Kronfeld, Jour. Am. Dent. Assn.)

BONY UNION (ANKYLOSIS) OF ROOT AND ALVEOLUS.

In spite of the close proximity between root surface and bone, a solid union of the two structures is very infrequent. Among several thousand human teeth that were sectioned *in situ*, in only two or three was spontaneous ankylosis between cementum and bone found. In animals, under certain experimental conditions, fusion between bone and tooth is more common. The high incidence of ankylosis in replanted teeth has already been mentioned. With the exception of replantation, chronic periapical infection is the most common cause

of ankylosis (Fig. 257). It causes extensive resorptions of the root, and when bone grows into the resorbed areas, it is likely to become united with the root. Another cause of a bony junction between root and jaw is experimental excessive occlusal trauma and subsequent relief. The trauma causes root resorption; later, these resorptions are repaired, and bone and tooth become united. Of similar origin also is the occasional union between deciduous teeth and bone (Fig. 236). Gottlieb and Orban found ankylosis in dogs' teeth following



FIG. 258.—Spontaneous bony union between teeth and alveolus in an old dog. BU, points of bony union. (Köhler, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

irritation of the periodontal membrane by experimental diathermy or by treatment of the root canal with formalin.

All of the above-mentioned instances of ankylosis have one thing in common: there was an injury to the periodontal membrane and the cementum that caused more or less extensive root resorption, and the bony union between alveolus and tooth developed while this root resorption was being repaired.

Köhler reported extensive bony ankylosis in the teeth of old dogs (Fig. 258). For some unknown reason the cementum had become extensively resorbed; subsequently, the bone grew toward the resorbed root surface and became united with it.

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CHAPTER XII.

EPITHELIAL ATTACHMENT AND GINGIVAL CREVICE.

BEFORE considering periodontal diseases, it is necessary to describe the normal conditions and physiological changes found in gingival tissues. Investigations in this field during the last two decades have resulted in many and important new findings that necessitate a revision of the earlier concept of the gingival tissues under normal and abnormal conditions.

EPITHELIAL ATTACHMENT.

The former concept of the relationship between tooth and gingivæ was that the gingivæ are attached to the tooth at the cemento-enamel junction. The soft tissues crownward from the cemento-enamel junction were called the free gingivæ. Investigations have shown that the condition just described does not exist and, consequently, all theoretical and practical considerations based on this concept had to be changed. The relationship between tooth surface and investing soft tissues will be described as it is found in specimens from human jaws of different ages.

Definition and Terminology.—In 1921, Gottlieb demonstrated that in a young tooth the bottom of the gingival crevice is not located, as was formerly believed, at the cemento-enamel junction but on the surface of the enamel, and that the cervical part of the enamel and the enamel epithelium are still united. Gottlieb called the epithelium that is in organic connection with the tooth surface the “epithelial attachment.”

The difference between the old and the new concept is illustrated by a diagram (Fig. 259).

Old Concept.—The epithelium is attached to the tooth in a line around the cemento-enamel junction (Fig. 259, left side at *c*).

New Concept.—The epithelium is not attached in a line but by a band. This band of epithelium (Fig. 259, right side *A-C*) surrounding the tooth is united with the tooth surface.

The epithelial attachment can be plainly seen in good tissue sections of jaws from men and animals. The location of the epithelial attachment on the tooth surface varies in different teeth, in different

individuals, and at different ages. But in every erupted tooth the oral epithelium is united with the tooth surface by an epithelial attachment.

In order to clarify the nomenclature in this particular field, the terms that are at the present time generally used will be enumerated here.

The term *gingival margin* is used to describe the free border or margin of the gingiva. The *crevice epithelium* extends from the gingival margin to the line of attachment of epithelium to the tooth surface; this line is the *bottom of the gingival crevice*. The *epi-*

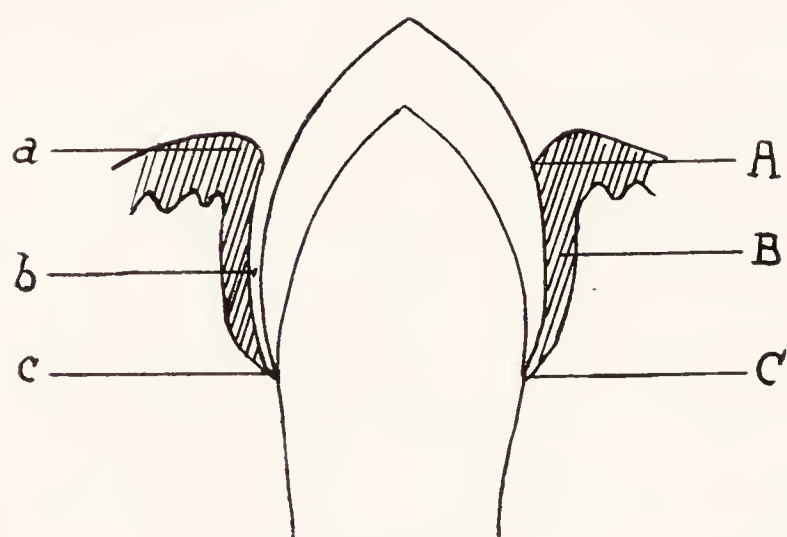


FIG. 259

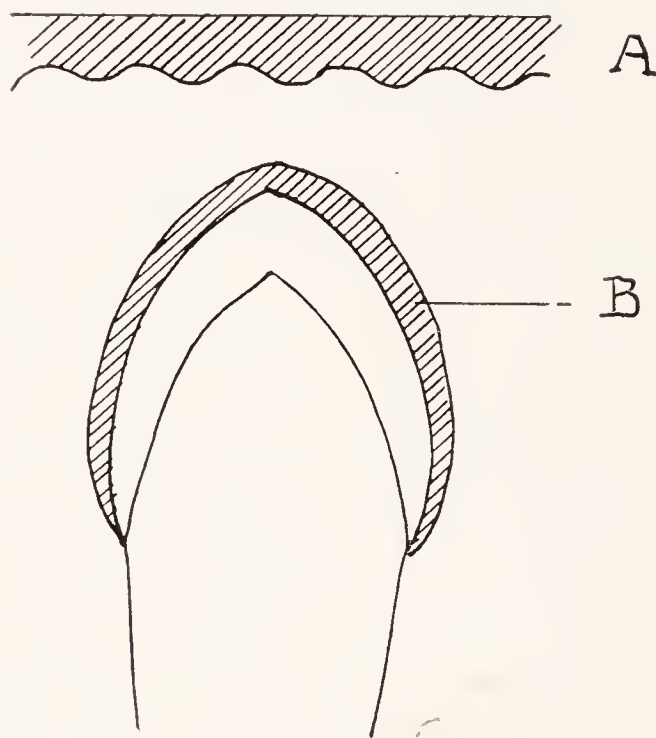


FIG. 260

FIG. 259.—Diagram illustrating the difference between the old and new concepts of the attachment of the gingival tissues to the tooth. The small letters are used to illustrate the old concept; the capitals, the new concept. *a*, free gingiva; *b*, subgingival space; *c*, gingival line; *A*, bottom of the gingival crevice; *B*, epithelial attachment; *C*, cemento-enamel junction (deepest point of the epithelial attachment).

FIG. 260.—Diagram illustrating a tooth before eruption. *A*, oral epithelium; *B*, enamel epithelium in organic connection with the enamel. (Orban and Mueller, Jour. Am. Dent. Assn.)

thelial attachment is that portion of the epithelium that is in organic connection with the tooth surface; it is found rootwise from the bottom of the crevice. The *gingival crevice* is the space that is bordered on one side by the tooth surface and on the other by the crevice epithelium.

Development of Epithelial Attachment and Gingival Crevice.—Epithelial attachment and gingival crevice can be fully understood only in connection with the tissue changes occurring during the eruption of the tooth.

The enamel of the tooth is formed by the enamel organ, which originally consists of four layers. The ameloblasts or ganoblasts,

cylindrical cells whose function it is to form the enamel, constitute the innermost layer of the enamel organ. After the enamel formation is completed, the ameloblasts disappear; the remaining layers of the enamel organ are fused and finally form several rows of stratified squamous epithelial cells, the reduced enamel epithelium. This layer of epithelial cells is in organic connection with the surface of the enamel; it separates the crown from the surrounding connective tissue of the jaw (Fig. 260). During the eruptive movement of the tooth, the crown moves closer to the oral epithelium, and on reaching it, the enamel epithelium on the crown surface unites with the oral epithelium (Fig. 261). Thus, when the tip of the enamel appears

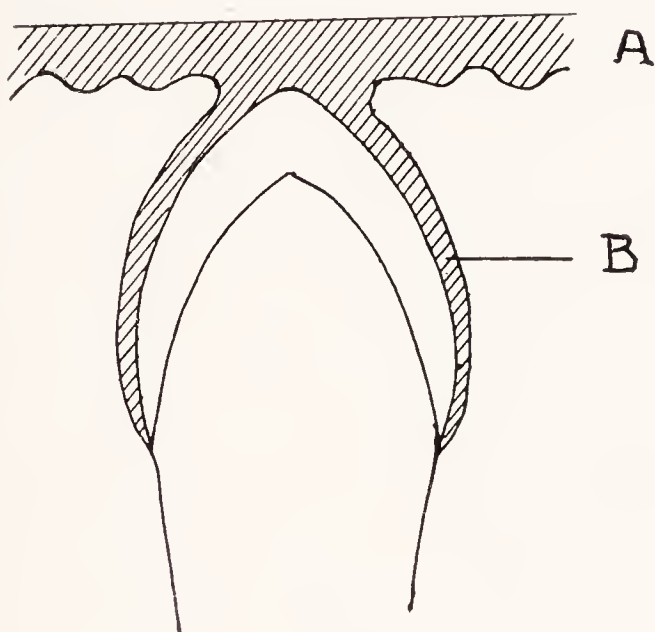


FIG. 261

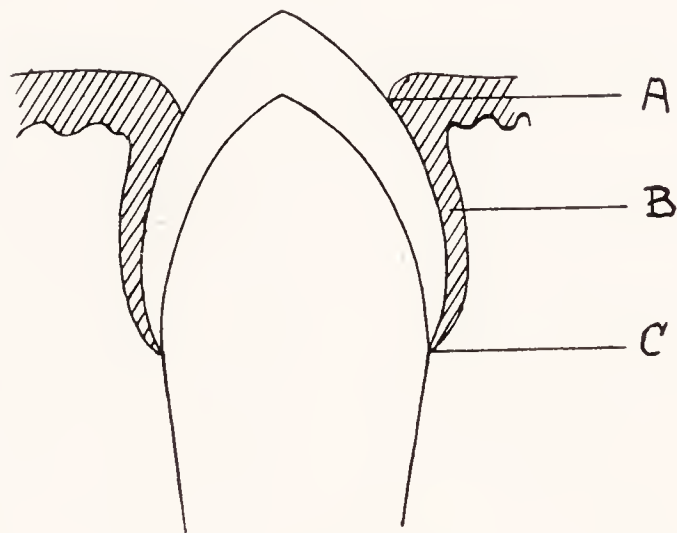


FIG. 262

FIG. 261.—The tooth is a little further advanced in eruption than in Fig. 260. *A*, oral epithelium; *B*, enamel epithelium. The epithelial tissues are united.

FIG. 262.—The tip of the enamel is erupted. *A*, bottom of the gingival crevice; *B*, epithelial attachment in organic connection with the enamel; *C*, cemento-enamel junction. (Orban and Mueller, Jour. Am. Dent. Assn.)

in the oral cavity, the oral epithelium is continuous with the enamel epithelium around the entire crown (Fig. 262). Since the enamel epithelium is in organic connection with the enamel surface, the cervical portion of the enamel is not exposed but is still united with the gingival tissue.

A drawing made from a specimen of an upper central incisor of a child, aged ten years, illustrates the actual condition at this period of life (Fig. 263). The crown has reached the occlusal plane and occludes with its antagonists. Approximately three-fourths of the enamel of the crown is exposed in the oral cavity; the gingival one-fourth is covered by the soft tissue that was formerly believed to be the "free gingivæ."

After it had been found that when the tooth reaches the occlusal plane the bottom of the gingival crevice is not at the cemento-enamel junction, it seemed advisable to introduce new terms for the erupted and the unerupted parts of the tooth surface. Every normal human tooth has a crown that is covered by enamel, and a root that is covered by cementum. Gottlieb called the enamel-covered portion of the tooth the “anatomical crown” and the cementum-covered portion the “anatomical root” (Fig. 264 *a*); between them is the

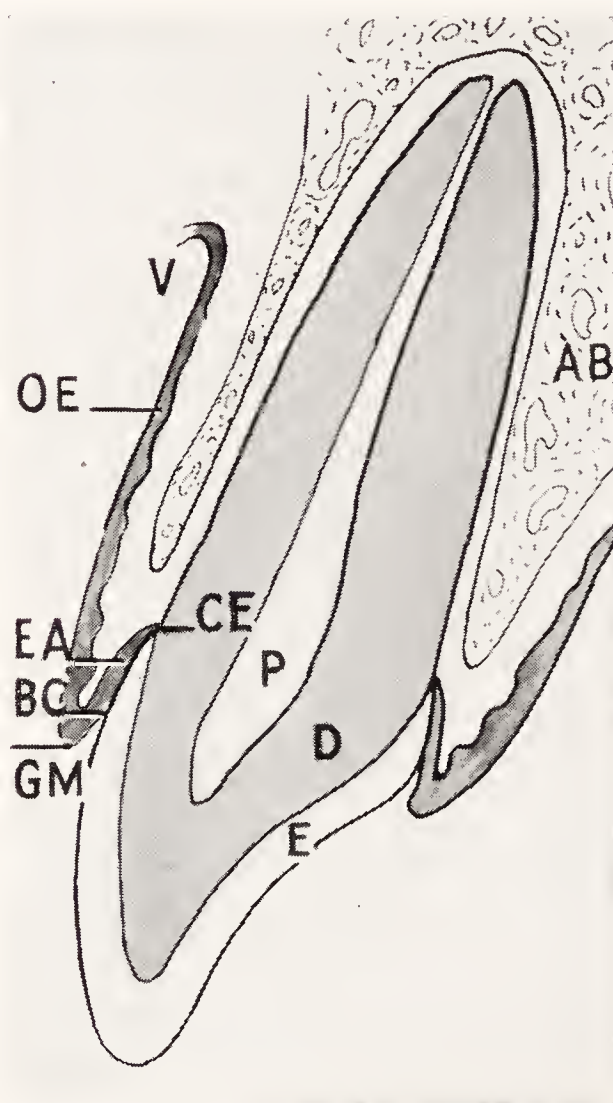


FIG. 263.—Diagram of an upper central incisor of a child at the age of ten years. Labio-lingual section. This diagram has been drawn from specimens, such as the one illustrated in Figures 265 and 266. *P*, pulp; *D*, dentin; *E*, enamel; *CE*, cemento-enamel junction; *OE*, oral epithelium; *V*, labial vestibule; *GM*, gingival margin; *BC*, bottom of the gingival crevice on the enamel surface; *EA*, epithelial attachment to the enamel; *AB*, alveolar bone. The free gingivæ extend from *BC* to *GM*.

cemento-enamel junction. The portion of the tooth that projects from the bottom of the gingival crevice is called the “clinical crown,” and the portion that extends rootward from the bottom of the gingival crevice the “clinical root” (Fig. 264, *b*, *c*, *d*). Clinical crown and clinical root can be distinguished only if the surrounding soft tissues are undisturbed; the relative size of clinical crown and clinical root depends upon the location of the bottom of the gingival crevice on the tooth surface.

Discovery of Epithelial Attachment.—There are several reasons why the existence of an epithelial attachment to the enamel was not discovered until rather recently and why some men engaged in dental research have not yet been able to convince themselves of its existence. These reasons are:

It is difficult to prepare histological sections of a tooth and the surrounding soft tissues and bone. In ground sections, most of the histological details of the soft tissues are destroyed. In decalcified sections of jaw specimens, misleading tears and distortions are likely to occur; the epithelial attachment is often torn away from its original position, making the correct interpretation of the specimens difficult and sometimes impossible.

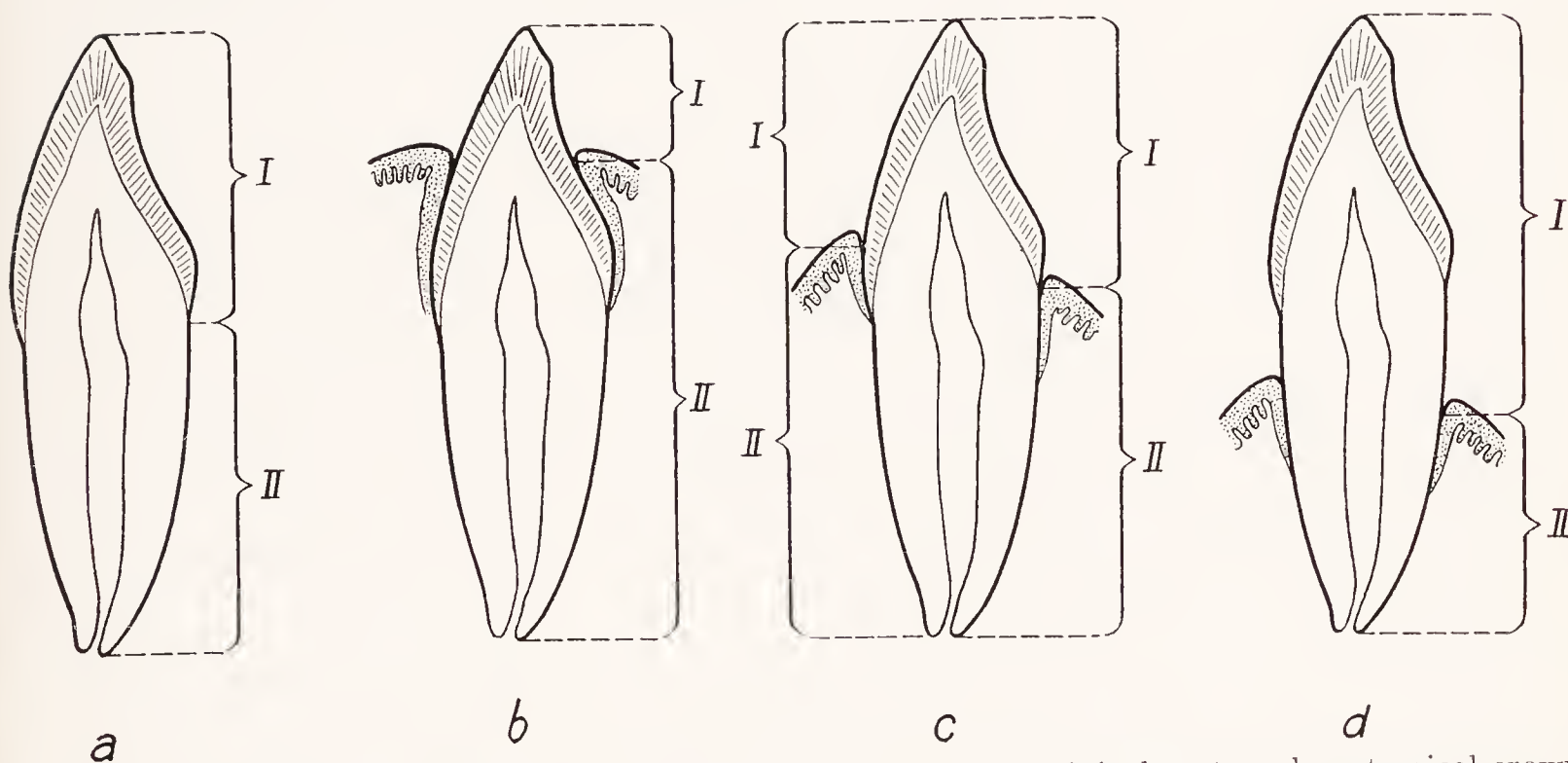


FIG. 264.—Diagram of the relations between clinical crown and clinical root, and anatomical crown and anatomical root, respectively. The anatomical crown is the enamel-covered part of the tooth (*a*, *I*); the anatomical root is the cementum-covered part of the tooth (*a*, *II*). The clinical crown is the part of the tooth which is above the bottom of the pocket *i*, *e.*, which projects into the oral cavity (*I* in *b*, *c*, *d*), the clinical root is the part of the tooth which is below the bottom of the pocket (*II* in *b*, *c*, *d*). In the course of the eruption of the tooth the clinical crown grows larger and larger, and the clinical root shorter and shorter. (Gordon, Dental Science and Dental Art.)

It is difficult to procure human jaw specimens for histological investigations. There are still dental scientists who are unable to obtain freshly fixed human jaw specimens of different ages for their studies. Without such specimens, however, a full understanding of the changes that occur in the gingival tissues during life is impossible. Some investigators have used animal tissues for their studies. However, the histological findings in human and animal specimens are not always identical or even analogous; therefore, the use of animal specimens is limited, unless they can be checked with similar human specimens.

Furthermore, it is necessary to decalcify teeth and bone in order to be able to section them on the microtome. In the process of decalcification the enamel is dissolved and it becomes difficult to visualize the anatomical relationships present before the loss of the enamel. In decalcified specimens the empty space crownward from the cemento-enamel junction between dentin and gingiva has been erroneously called the "gingival space"; in reality this is not the gingival space but the space formerly occupied by the enamel.

It is sometimes difficult to distinguish between pathological and normal conditions. Many specimens heretofore used in text-books and publications to illustrate the relationship between gingival tissues and tooth surface showed definitely pathological changes, such as extensive destruction of the epithelial attachment by inflammatory processes. Such specimens cannot be used to illustrate the normal relationship between tooth and gingiva.

For these reasons the earlier investigators did not know about the epithelial attachment. Gottlieb gave the first description of it in 1921, and in an article by Prinz (1926), these findings were first mentioned in American literature. Skillen and Mueller were the first ones in the United States to give a correct description of gingival crevice and epithelial attachment, using their own histological specimens (1927). But now this knowledge of the epithelial attachment has found its way into most of the modern text-books of dental and general histology.

MICROSCOPIC APPEARANCE OF THE GINGIVAL CREVICE.

How is it possible to determine in decalcified specimens of human teeth and jaws where the bottom of the gingival crevice was located during life? Several landmarks make it possible to interpret correctly specimens of this kind:

Location of the enamel cuticle (Nasmyth's membrane).

Location of the subepithelial infiltration.

Difference in appearance of epithelial attachment and crevice epithelium.

Location of calculus in the crevice.

Location and Significance of the Enamel Cuticle.—The enamel cuticle or Nasmyth's membrane is a thin layer of organic tissue that is attached to the surface of the enamel. In young people in whom only a part of the enamel is erupted, the cuticle covers the exposed portion of it. After the enamel has been dissolved in the preparation of the specimen, the part of the cuticle crownward from the bottom

of the gingival crevice is left and is visible in the specimen as a fine thread extending from the bottom of the gingival crevice in the direction of the former surface of the enamel.

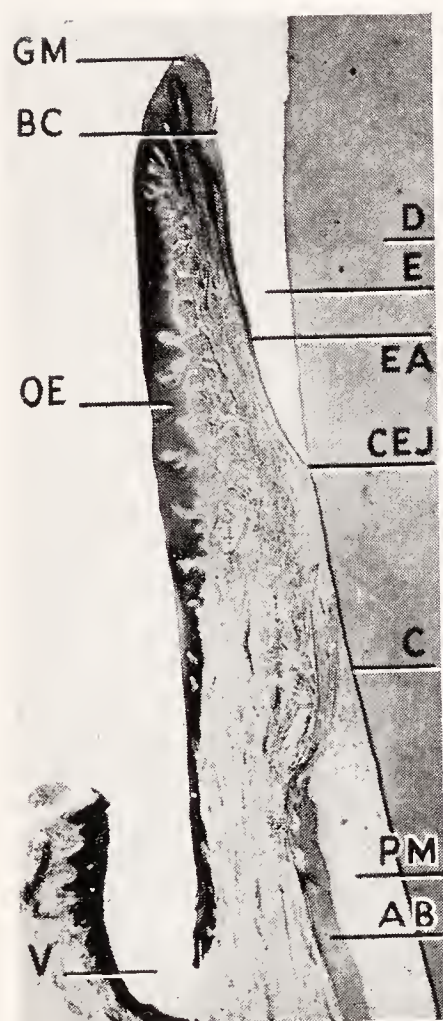


FIG. 265

FIG. 265.—Photomicrograph of one of the specimens that were used in drawing the diagram, Figure 263. Labial side of an incisor of a child, aged ten years. *D*, dentin; *E*, enamel space; *CEJ*, cemento-enamel junction; *EA*, epithelial attachment; *BC*, bottom of gingival crevice; *GM*, gingival margin; *GM-BC*, gingival crevice; *OE*, oral epithelium; *V*, labial vestibule; *C*, cementum; *AB*, alveolar bone; *PM*, periodontal membrane.

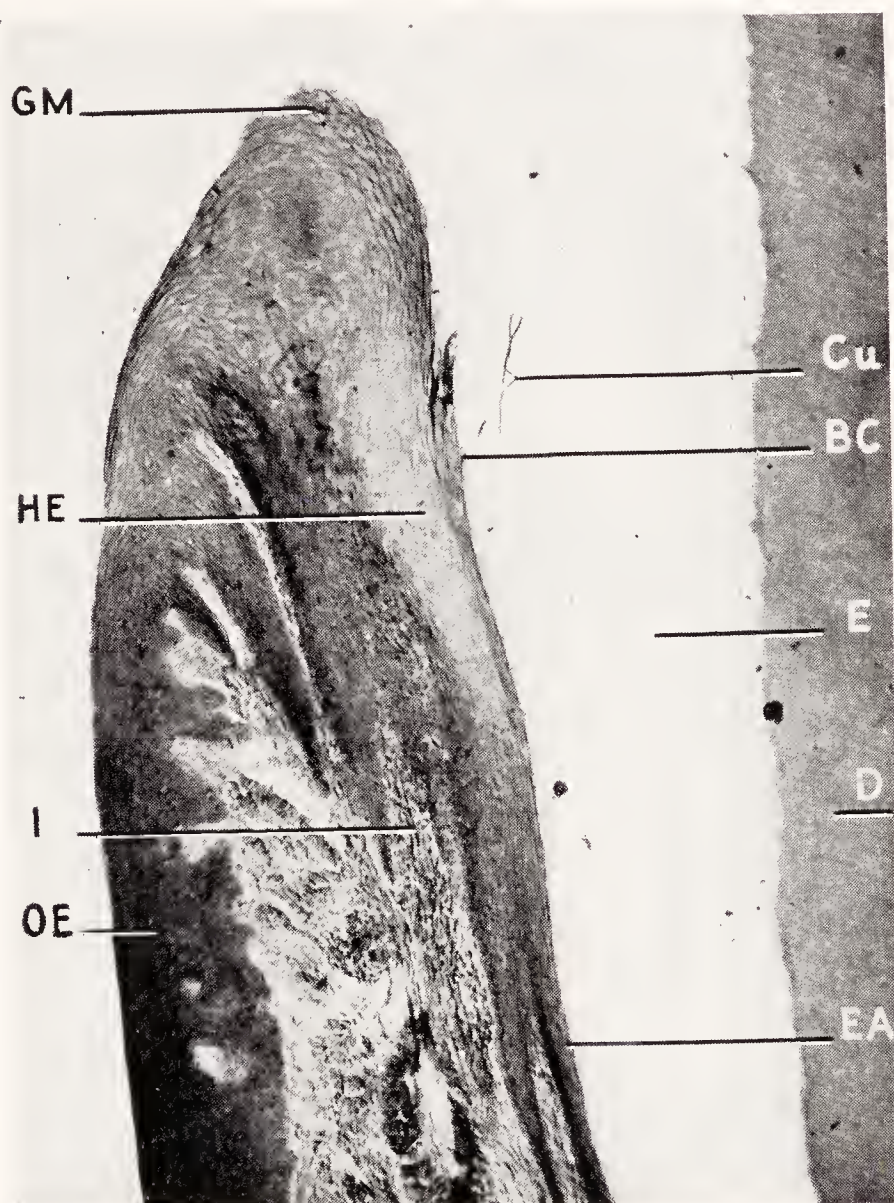


FIG. 266

FIG. 266.—Higher magnification of the gingival margin in Figure 265. *D*, dentin; *E*, enamel space; *EA*, epithelial attachment to the enamel; *Cu*, enamel cuticle; *BC*, bottom of the gingival crevice; *HE*, incipient hornification of the cells of the epithelial attachment initiating the process of detachment of the epithelium from the enamel surface; *GM*, gingival margin; *OE*, oral epithelium; *I*, subepithelial round-cell infiltration.

Figure 265 shows one of the sections that was used in drawing the diagram, Figure 263. It represents the labial side of an upper incisor of a child with clinically normal, healthy gingivæ. The enamel has been lost in the preparation of the specimen; the outline of its cervical portion is shown by the epithelial attachment, *EA*.

Toward the gingival margin the epithelium of the epithelial attachment becomes thicker; in a higher magnification of the gingival margin (Fig. 266) it can be seen that the epithelium has changed: the cells are lighter and larger and show signs of the beginning of hornification. At *BC*, the cuticle is detached from the inner surface of the epithelial attachment, indicating the location of the bottom of the gingival crevice. The gingival crevice extends from *BC* to *GM*; its depth is about 0.03 mm., or practically zero. The cuticle, which followed the outline of the enamel surface before decalcification, lost its support when the enamel dissolved, and became folded.

According to Gottlieb, there are two types of cuticle, the primary and the secondary. The primary cuticle, the last product of the ameloblasts, is formed on the enamel surface before the ameloblasts degenerate and disappear. It is about 1 micron thick and usually becomes calcified. It often disappears when the enamel is decalcified. The secondary cuticle is a structure formed by the squamous cells of the epithelial attachment. It is homogeneous, appears bright pink when stained with eosin, and is about 7 microns thick. The secondary cuticle is on the enamel as well as on the root surface and, therefore, has been called *cuticula dentis*. It is resistant to alkalis and acids; therefore, it withstands decalcification during the preparation of histological sections, and is frequently seen in the latter.

Location of the Subepithelial Infiltration.—Another important aid in localizing the bottom of the gingival crevice is the position of the subepithelial infiltration. The great majority of microscopic specimens of gingival crevices in man as well as in animals shows round-cell infiltration in the tissue immediately below the bottom of the gingival crevice.

In Figure 266 this round-cell infiltration is located at *I*, corresponding to the location of the bottom of the gingival crevice at *BC*. A high magnification of the cemento-enamel junction of this specimen shows no trace of inflammatory infiltration (Fig. 267).

Difference in Appearance of Epithelial Attachment and Crevice Epithelium.—The epithelial attachment consists of several layers of stratified squamous epithelial cells. The side that faces the enamel has the same outline and curvature as the enamel surface to which it was attached.

The crevice epithelium is usually thicker than the epithelial attachment and is characterized by the presence of high papillæ in the subepithelial connective tissue. The surface of the crevice epithelium forms the wall of the gingival crevice, and its outline depends entirely upon the condition of the crevice; therefore, it

never appears as even and uniform as the epithelial attachment. On the distal side of a cuspid (Fig. 268), the distance from the gingival margin to the cemento-enamel junction was measured and found to be 3.5 mm. How much of this distance is actually the depth of the gingival crevice? A higher magnification of the area around the cemento-enamel junction shows that the epithelium from *CEJ* to *BC* follows in a smooth curve the original outline of the enamel surface; at *BC* is the enamel cuticle, indicating that *BC* is the bottom of the gingival crevice; consequently, the epithelium between this point

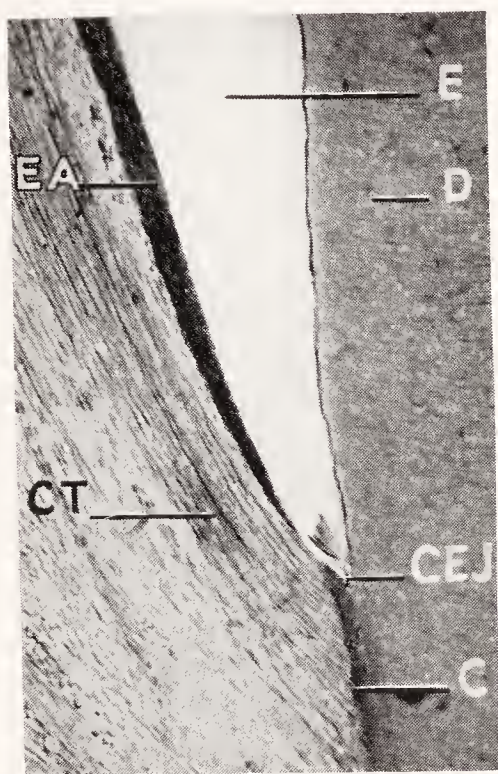


FIG. 267.—High magnification of the cemento-enamel junction in Figure 265. *D*, dentin; *E*, enamel space; *CEJ*, cemento-enamel junction; *EA*, epithelial attachment to the enamel; *CT*, connective tissue; *C*, cementum. Notice the absence of round-cell infiltration in the subepithelial connective tissue.

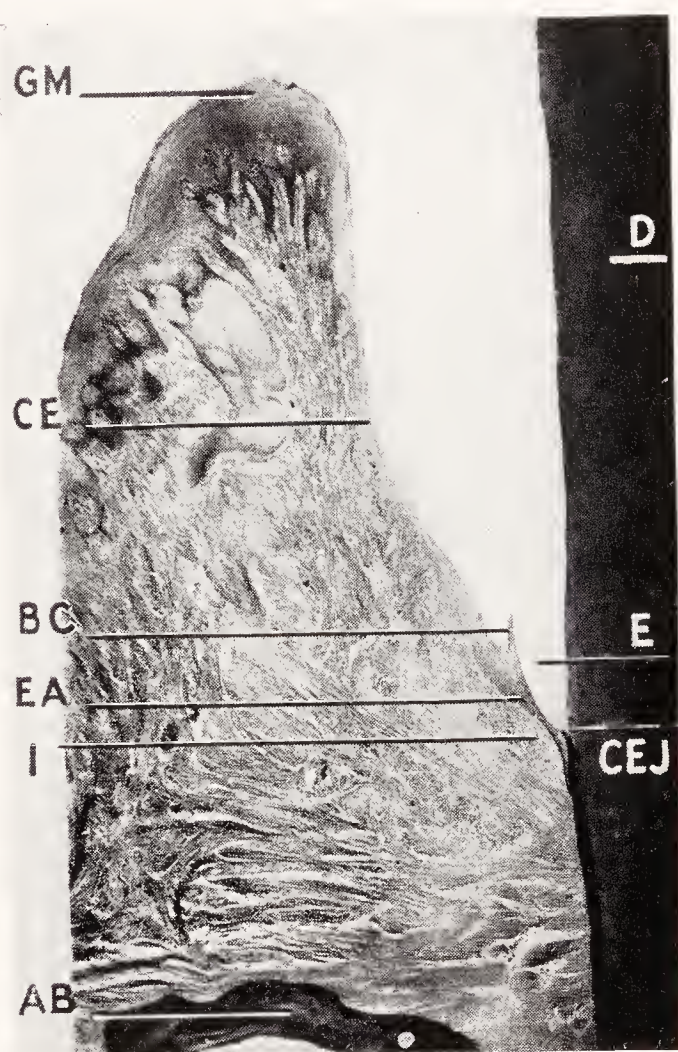


FIG. 268.—Gingival crevice on the distal side of a lower cuspid. *D*, dentin; *E*, enamel space; *CEJ*, cemento-enamel junction; *EA*, epithelial attachment to the enamel; *BC*, bottom of the gingival crevice; *CE*, crevice epithelium; *I*, subepithelial round-cell infiltration; *GM*, gingival margin; *AB*, alveolar bone. The gingival crevice extends from *GM* to *BC*; its depth is 3 mm.; the length of the epithelial attachment to the enamel (*BC* to *CEJ*) is 0.45 mm.

and the cemento-enamel junction is the epithelial attachment. This location of the bottom of the crevice is corroborated by the location of the subepithelial infiltration. The gingival crevice extends from *BC* to *GM*.

Location of Calculus in the Crevice.—Calculus is present in most of the gingival crevices in human jaw specimens. Since calculus is

a product of the saliva, it can be deposited only on that part of the tooth surface that is accessible to the oral fluids. The most apical extension of calculus found in any section of a gingival crevice indicates the location of the bottom of the crevice.

SIGNIFICANCE OF THE TERM EPITHELIAL ATTACHMENT.

The presence of an epithelial attachment to the enamel is still doubted by some. But as an increasing number of investigators succeeds in preparing good histological sections of human and animal teeth, the existence of such an organic connection between tooth surface and epithelium becomes more generally known. The way in which the epithelial cells are fastened to the tooth surface is not yet fully understood. Manley gives the following explanation: The primary cuticle is continuous with the organic substance of the prism sheaths of the enamel. During the process of eruption the primary cuticle fuses with the hornified layer of the oral epithelium. This organic junction between oral epithelium, secondary and primary enamel cuticles, and prism sheaths constitutes the epithelial attachment to the enamel.

Whether the epithelial attachment is "attached" to the enamel or merely in "close contact" with it can be determined by a study of the histological findings in extracted teeth. During extraction the tissues are torn from the body where there is the least resistance. In examining extracted teeth from young people, the epithelial attachment is frequently still connected to the enamel surface, indicating that the connection between tooth surface and epithelium was stronger than that between epithelium and underlying tissue.

Figures 269 and 270 show such a specimen. The photomicrographs were taken from the mesial side of a lower bicuspid that was extracted because of pulpitis. The extracting forceps grasped the tooth bucco-lingually; therefore, the area reproduced in these illustrations was not touched by the forceps, and any soft tissue found in this location must have been attached to the tooth surface. The mesial side near the cemento-enamel junction is covered by a thin layer of soft tissue extending over the enamel as well as over the cementum. In a higher magnification it can be recognized as stratified squamous epithelium. This shows that the mechanical attachment of the epithelium to enamel and cementum was strong enough to withstand the laceration of the tissues during extraction.

The statement is sometimes made that it is possible to pass an explorer between the enamel and the gingival tissues of young

patients. However, if this is done gently, it is always possible to feel a definite resistance—the bottom of the gingival crevice—before the explorer reaches the cemento-enamel junction. Of course, one can force the explorer between enamel surface and epithelial attachment and thus detach the latter; but this is not proof that an attachment did not exist before the exploration.

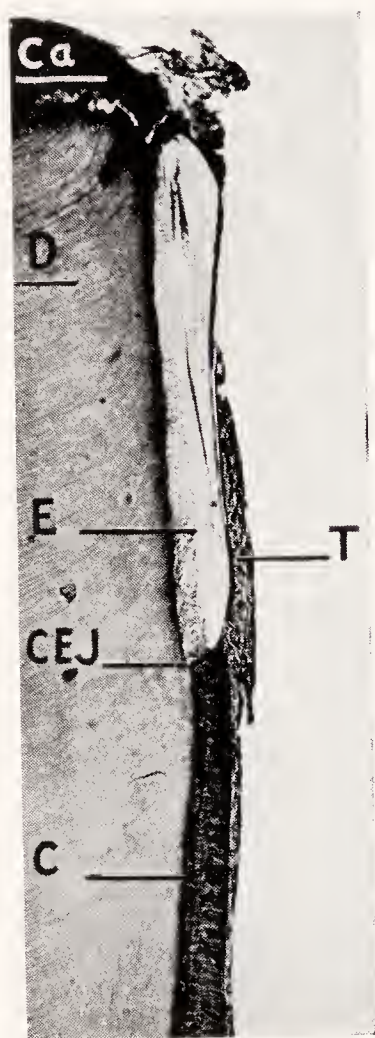


FIG. 269

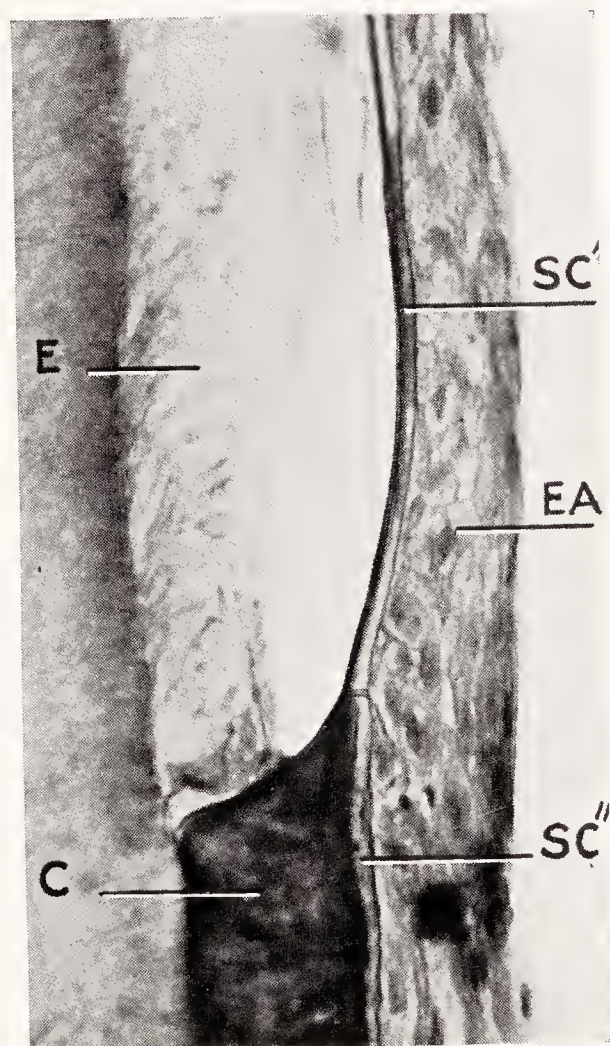


FIG. 270

FIGS. 269 and 270.—The attachment of the epithelium to the enamel and cementum.

FIG. 269.—Cemento-enamel junction of a human lower bicuspid. The tooth was extracted, decalcified, and embedded in celloidin. *D*, dentin; *E*, enamel space; *C*, cementum; *CEJ*, cemento-enamel junction; *Ca*, caries; *T*, soft tissue attached to the enamel and to the cementum.

FIG. 270.—Higher magnification of the cemento-enamel junction. *E*, enamel space; *C*, cementum; *SC'*, hornified secondary cuticle on the enamel; *SC''*, hornified secondary cuticle on the cementum; *EA*, stratified squamous epithelium of the epithelial attachment. This epithelium was torn from the subepithelial connective tissue during extraction of the tooth and remained attached to the tooth surface. (Kronfeld, Jour. Am. Dent. Assn.)

In summarizing, the following description can be given of the relationship between gingival soft tissues and tooth surface in young persons: At the time when the tooth reaches the occlusal plane, the erupted part of the enamel, the clinical crown, includes two-thirds to three-fourths of the length of the crown; the cervical por-

tion of the enamel is still in organic connection with the epithelial attachment. The bottom of the gingival crevice is located on the enamel surface at the border-line between crevice epithelium and epithelial attachment. The gingival crevice extends from this point crownward to the free gingival margin; the wall of the gingival crevice is formed by the crevice epithelium on one side and by the enamel or the enamel cuticle on the other.

CLINICAL SIGNIFICANCE OF THE EXISTENCE OF AN EPITHELIAL ATTACHMENT TO THE ENAMEL.

Since in young individuals the cervical part of the enamel is in organic junction with the epithelial attachment, this part of the crown is not exposed to the fluids of the oral cavity and, therefore, cannot decay. This observation was described by G. V. Black. He showed specimens of teeth of young individuals with extensive approximal and cervical decay in which the gingival portion of the enamel had remained intact. Black did not know about the existence of an epithelial attachment to the enamel; he thought that the bottom of the gingival crevice was at the cemento-enamel junction. Consequently, he drew the only logical conclusion, namely, that the gingival crevice is a self-cleansing area and that the enamel is protected from caries by the free margin of the gingiva. From the findings described in the foregoing paragraphs, it is clear that the presence of smooth, intact enamel in the gingival portion of young teeth must be explained differently. The cervical portion of the enamel is not protected by the gingiva but is still united with it by means of the epithelial attachment; therefore, it is inaccessible to bacteria and cannot decay. In a tooth like the one shown in Figures 263 and 265, only the portion of the enamel crownward from *BC* (clinical crown) is accessible to caries. The enamel from *BC* to *CEJ* has not yet erupted. If the tooth were extracted, the cervical portion of the enamel would be intact even if there were extensive gingival or approximal decay.

Another important clinical observation is the relationship between epithelial attachment and deposits of calculus. Calculus can be deposited only on the surface of the clinical crown. Since the bottom of the gingival crevice in young individuals is located on the enamel surface, no calculus can be deposited on the enamel rootward from the bottom of the crevice. If such a tooth is extracted, calculus is frequently found deposited in a more or less complete ring around the cervical portion of the enamel. Between the calculus and the

cemento-enamel junction there is a band of clean, white enamel without any deposit on it. In correlating this clinical observation with the microscopic findings in young teeth, it can easily be understood that the white area of enamel rootward from the deposit corresponds to the area between *BC* and *CEJ*, which was not yet erupted.

LOCATION OF GINGIVAL CREVICE AND EPITHELIAL ATTACHMENT ON THE TOOTH SURFACE AT DIFFERENT PERIODS OF LIFE.

When a tooth reaches the occlusal plane, about one-third or one-fourth of the enamel is still united with the epithelial attachment. Rootward, the epithelial attachment ends at the cemento-enamel junction. This location, however, is not permanent. Gradually, more and more of the enamel is exposed by a separation of the epithelial attachment from the tooth surface. At the same time, the tissue of the free gingivæ is reduced by atrophy so that an increasing amount of enamel is exposed. The eruption of the tooth continues, although at a very much slower rate than in the first years of eruption. As a result of this continued detachment, the bottom of the gingival crevice gradually approaches the cemento-enamel junction.

Proliferation of the Epithelial Attachment Along the Cementum.—Before the bottom of the gingival crevice reaches the cemento-enamel junction, another process invariably takes place: the deepest point of the epithelial attachment at the cemento-enamel junction proliferates and grows along the cementum of the root (Fig. 271). Thus, when the bottom of the gingival crevice reaches the cemento-enamel junction, the deepest point of the epithelial attachment is already located on the cementum.

Figure 272 illustrates this condition. The bottom of the gingival crevice is located at *BC*, on the enamel surface. The deepest point of the epithelial attachment has passed the cemento-enamel junction and is found on the cementum. The epithelial attachment, therefore, can be divided into an epithelial attachment to the enamel (*BC* to *CEJ*) and an epithelial attachment to the cementum (*EA''*). The subepithelial connective tissue is infiltrated with inflammatory exudate cells (polyblasts). The depth of the crevice is zero.

As epithelium and enamel surface continue to separate, the bottom of the gingival crevice finally reaches the cemento-enamel junction (Fig. 273). At this stage, the bottom of the gingival crevice and the cemento-enamel junction coincide. The epithelial attachment extends from the cemento-enamel junction rootward along the surface

of the cementum. The epithelial lining of the gingiva is intact; the crevice has no depth.

Microscopic Structure of the Epithelial Attachment.—The epithelial attachment consists of the typical stratified squamous epithelial cells that compose the oral epithelium. Two sides of the epithelial attachment can be distinguished: the inner side, connected with the subepithelial connective tissue, and the outer side, facing

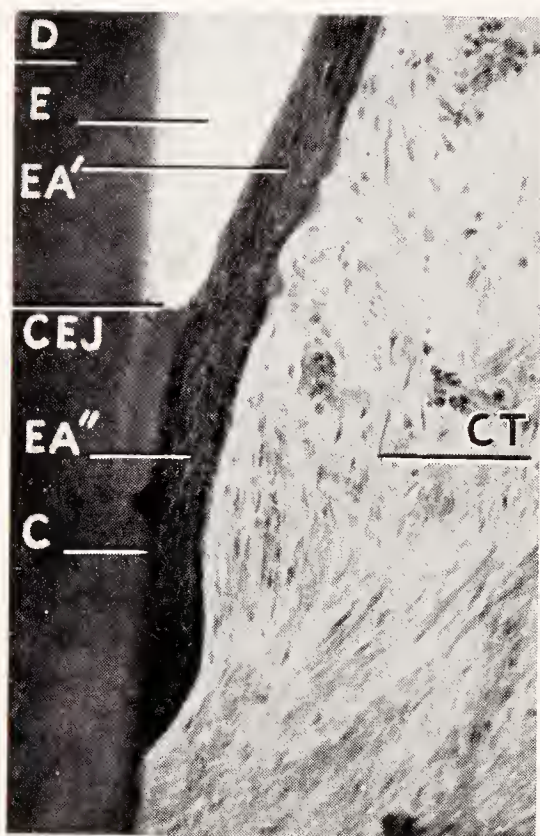


FIG. 271.—Proliferation of the epithelial attachment along the cementum. *D*, dentin; *E*, enamel space; *C*, cementum; *CEJ*, cemento-enamel junction; *EA'*, epithelial attachment to the enamel; *EA''*, epithelial attachment to the cementum; *CT*, subepithelial connective tissue, free of inflammation.

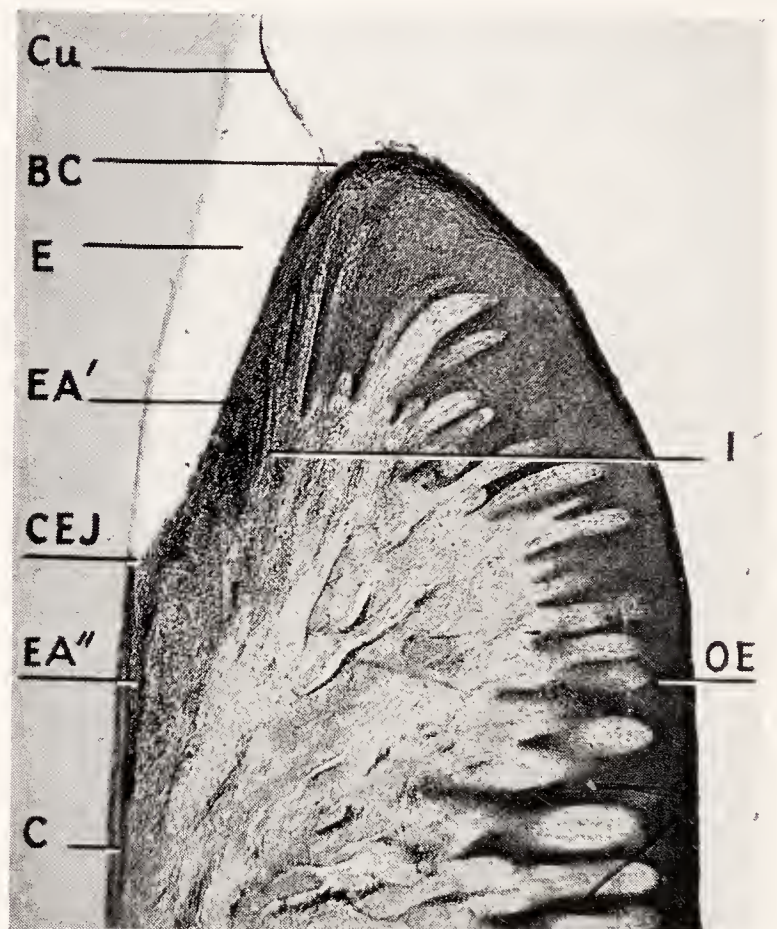


FIG. 272.—The deepest point of the epithelial attachment has passed the cemento-enamel junction and is on the cementum. The bottom of the gingival crevice is still located on the enamel. *E*, enamel space; *C*, cementum; *CEJ*, cemento-enamel junction; *EA''*, epithelial attachment to the cementum; *EA'*, epithelial attachment to the enamel; *BC*, bottom of the gingival crevice; *I*, round-cell infiltration; *Cu*, cuticle; *OE*, oral epithelium.

the tooth and attached to the tooth surface. The terms inner and outer side have been taken over from the gingival epithelium, the inner side of the gingival epithelium being the germinative or basal layer where new cells are formed, and the outer side being the hornified surface toward the oral cavity. Since the epithelial attachment is a duplicature or invagination of the oral epithelium around the tooth, the hornified cuticle on the tooth surface corresponds to the hornified layer on the surface of the gingival epithelium.

Orban described the form and the distribution of the cells of the epithelial attachment. He showed that the cells forming the epithelial attachment to the cementum are not different from those forming the attachment to the enamel (Fig. 274). In both cases the inner surface of the epithelial attachment is formed by a layer of regular, cuboidal cells with round, dark nuclei, which are the basal cells of the epithelium. Toward the tooth surface these basal cells



FIG. 273

FIG. 273.—Bottom of the gingival crevice at the cemento-enamel junction. *D*, dentin; *C*, cementum; *CEJ*, cemento-enamel junction; *BC*, bottom of gingival crevice; *Cu*, cuticle; *EA*, epithelial attachment to the cementum; *OE*, oral epithelium. The depth of the crevice is zero.



FIG. 274

FIG. 274.—High magnification of the epithelial attachment at the cemento-enamel junction. *D*, dentin; *C*, cementum; *E*, enamel space; *CEJ*, cemento-enamel junction; *Cu'*, hornified (secondary) cuticle on the enamel; *Cu''*, hornified cuticle on the cementum; *FE*, flat epithelial cells with intercellular bridges; *CE*, cuboidal epithelial cells in the basal layer of the epithelial attachment; *CT*, subepithelial connective tissue. (Orban, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

are covered by several layers of flat, squamous cells that are connected with each other by intercellular bridges. Next to the cementum, the nuclei are irregular and light; directly upon the cementum is a hornified cuticle. Toward the cemento-enamel junction, the cuticle becomes gradually thicker; at the junction it is continuous from the cementum to the enamel. The arrangement of the cells in the epithelial attachment to the enamel is the same as on the surface of the cementum: a regular, basal cell layer next to the subepithelial

connective tissue, flat cells arranged parallel to the tooth surface, and a layer of cells with small, irregular nuclei next to the cuticle.

Further crownward on the enamel surface, close to the bottom of the gingival crevice, the epithelial attachment shows certain characteristic changes. The cells begin to degenerate, thereby preparing the way for the separation of the epithelial attachment from the cuticle (Fig. 275). The epithelial cells next to the bottom of the

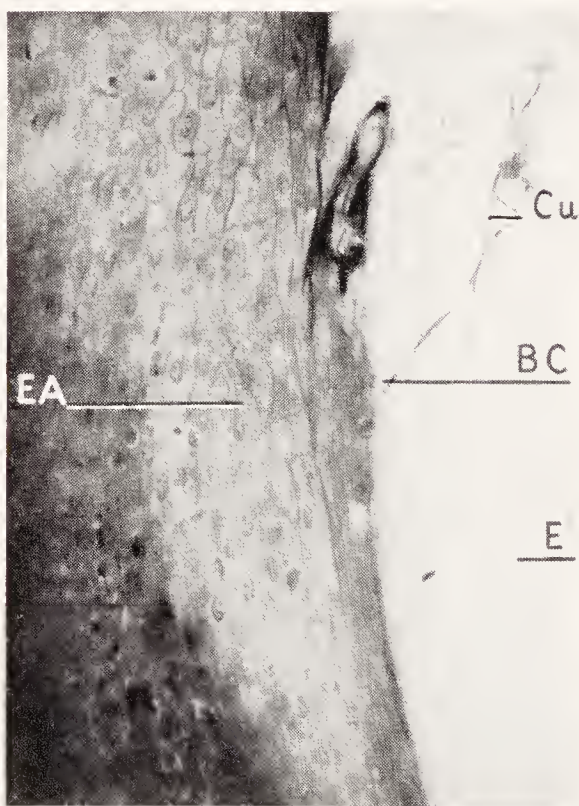


FIG. 275.—High magnification of the bottom of the gingival crevice. Degeneration of the superficial cells of the epithelial attachment and detachment of the cuticle. *E*, enamel space; *BC*, bottom of gingival crevice; *Cu*, enamel cuticle; *EA*, large, light, hornifying cells of the epithelial attachment next to the bottom of the crevice.

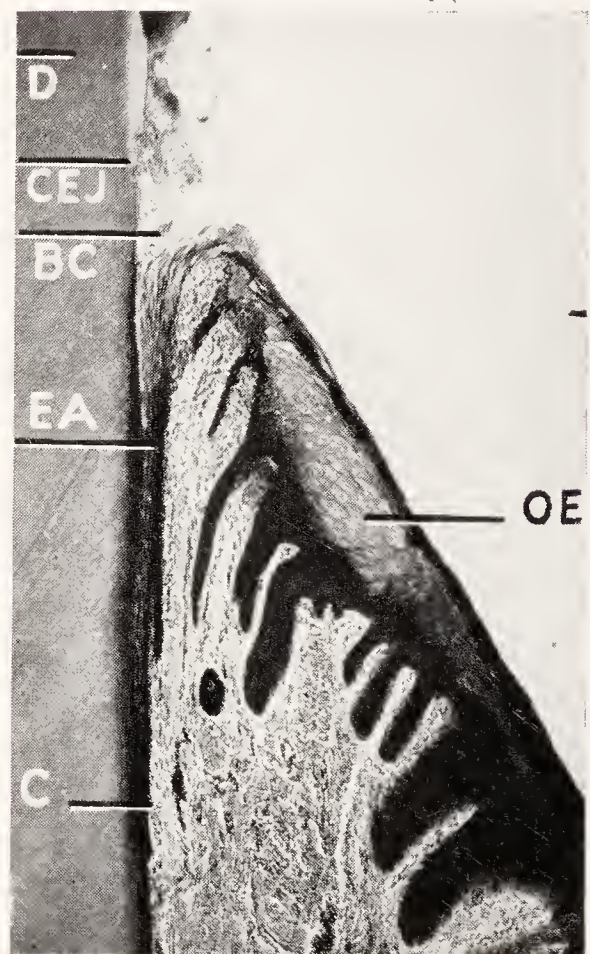


FIG. 276.—Bottom of the gingival crevice on the surface of the cementum. Labial surface, upper first bicuspid. *D*, dentin; *C*, cementum; *CEJ*, cemento-enamel junction; *BC*, bottom of the gingival crevice; *EA*, epithelial attachment to the cementum; *OE*, oral epithelium. The depth of the crevice is zero. (Kronfeld, Jour. Am. Dent. Assn.)

gingival crevice are pale and irregular; their nuclei are shrunken and frequently have disappeared. From these findings it is evident that the gingival crevice is formed by a degeneration of those cells of the epithelial attachment that are lying next to the tooth surface, and by their detachment from the cuticle.

Gingival Crevice and Cemento-enamel Junction.—The cemento-enamel junction has no significance whatsoever in the detachment of the epithelial attachment from the tooth surface. The location of the

bottom of the crevice at this point is a transitory stage in the course of the gradual separation of the soft tissues from the tooth surface. In the microscopic examination of a large number of human teeth of different ages, the bottom of the gingival crevice is only rarely found at the cemento-enamel junction; even in the few sections in which it has been observed at this point, the neighboring sections through the same tooth showed it either slightly more crownward or rootward from the cemento-enamel junction.

Later in life, the bottom of the gingival crevice is normally located on the surface of the cementum. The crevice may be extremely shallow, especially if the mouth is well cared for. The subepithelial connective tissue may be free of cellular infiltration (Fig. 276).

The changing relationship between epithelial attachment and tooth surface is illustrated in a diagram (Fig. 277). Figure 277, *a*, shows the relationship between tooth germ and jaw epithelium. The enamel is covered with enamel epithelium. In Figure 277, *b*, the erupting tooth has reached the jaw surface, and enamel epithelium and jaw epithelium have fused. Figure 277, *c*, shows the tip of the enamel erupted; most of the enamel epithelium is still united with the enamel, forming the epithelial attachment. In Figure 277, *d*, the epithelial attachment on the labial (left) side of the tooth terminates at the cemento-enamel junction; on the lingual (right) side it has proliferated rootward past the cemento-enamel junction. On the labial (left) side in Figure 277, *e*, the bottom of the crevice is at the cemento-enamel junction, while on the lingual (right) side it is on the enamel surface. Finally, Figure 277, *f* and *g*, show the adult stages of tooth eruption. The bottom of the crevice is on the root surface. The tooth is worn down, but at the same time it extrudes from the socket, and cementum has been laid down in thick layers at the apex.

In teaching the subject of the gingival crevice, the author has observed that the student easily makes the mistake of confusing "downward growth of the epithelial attachment" with "deepening of the crevice." These two terms are not synonymous. The shifting toward the apex of the bottom of the gingival crevice may continue all during life without ever being accompanied by a deepening of the crevice. This is especially true of mouths in good hygienic condition. In such mouths, crevices of zero depth can frequently be observed; in youth, they are located on the surface of the enamel, in advanced age, on the surface of the cementum. In some of the microscopic specimens reproduced in this book, attention has been called to such extremely shallow crevices in different locations on the tooth surface.

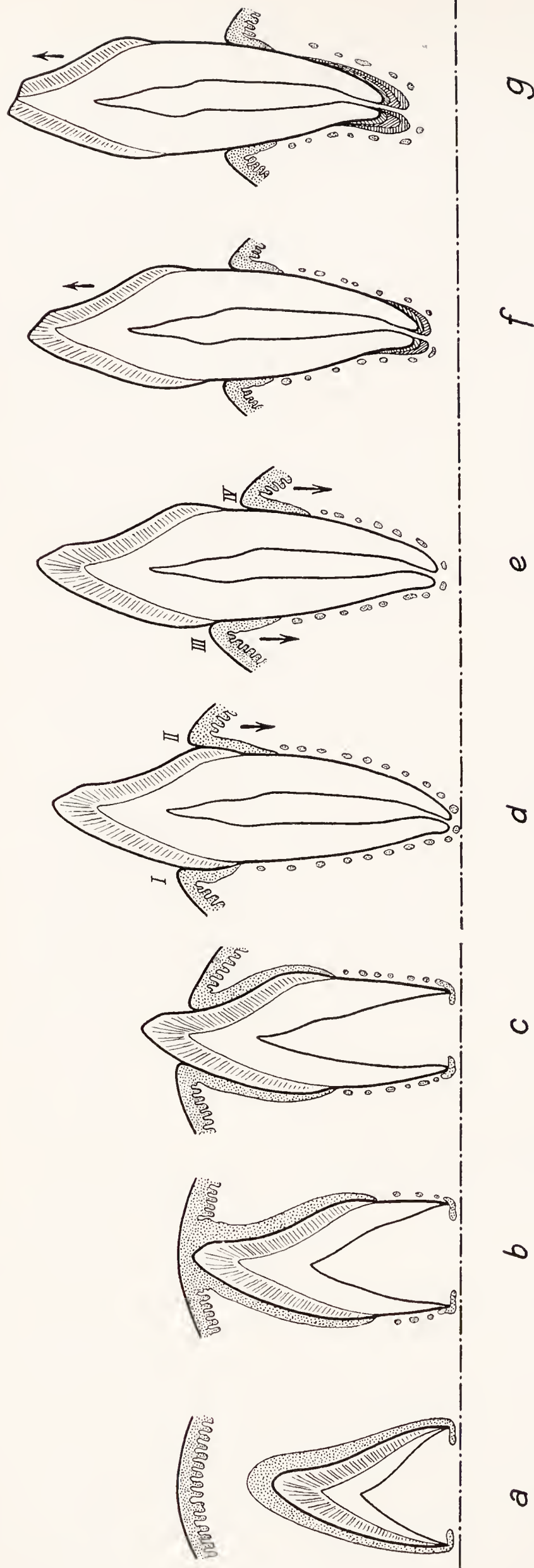


FIG. 277.—DIAGRAM EXPLAINING THE ERUPTION OF THE TOOTH.

(a) Tooth germ before eruption. The enamel-covered coronal part is covered with epithelium. The epithelium ends at Hertwig's sheath.

(b) The tip of the crown has reached the oral epithelium. Oral epithelium and enamel epithelium have coalesced.

(c) The tip of the crown has penetrated the epithelium; the oral epithelium is continuous with the former enamel epithelium, now called epithelial attachment, and extends as far as the cemento-enamel junction. The epithelial attachment extends from the bottom of the crevice, *i. e.*, the place where the epithelium is connected with the tooth surface, to the cemento-enamel junction.

(d) With the continuous development of the root the tooth erupts farther into the oral cavity, that is, the part of the tooth which freely projects into the mouth becomes bigger and bigger, the epithelial attachment shorter and shorter; but the epithelial attachment still extends as far as the cemento-enamel junction (*I*) and the bottom of the crevice is still connected with the enamel, *i. e.*, all the enamel covered part of the tooth has not yet erupted into the oral cavity. At the further progress of the eruption the epithelial attachment, which up until now had extended as far as the cemento-enamel junction, proliferates apeward along the root surface (*II*). The bottom of the crevice still touches the enamel. As the tooth erupts farther and farther, the epithelium grows farther and farther apeward along the cementum, the crevice bottom is also shifted apeward, and during this process the bottom of the crevice passes the cemento-enamel junction. *i. e.*, the whole enamel covered part of the tooth now projects into the oral cavity (*III*). It should be noted here that this state never occurs all around the circumference of a tooth at the same time; at some places it is not yet reached, while at others the bottom of the crevice has already receded as far as the cementum, so that part of the cementum projects into the oral cavity (*IV*).

This apeward proliferation of the epithelium from the dental surface progresses continuously, in one case more slowly, in another case more quickly (*f-g*). The tooth shows an active rise out of the alveolus. Stages *a* and *b* may be classified as the pure form of the active eruption of the tooth, while the further stages *c-g* represent a combined form of the active eruption of the tooth, *i. e.*, of the active eruption towards the plane of occlusion, and of the passive eruption of the epithelium from the tooth surface and the continuous apeward proliferation of the epithelium along the cementum. (Gordon, Dental Science and Dental Art.)

SIGNIFICANCE OF THE GRADUAL DETACHMENT OF THE GINGIVAL SOFT TISSUES FROM THE TOOTH SURFACE.

The separation of epithelial attachment from the root surface does not proceed at a uniform rate of speed. It is rapid in the first years after the tooth has started to erupt; later it is much slower. The rate of detachment, clinically known as recession, is subject to great individual variations. In some mouths, the cervical portion of the roots may be exposed at a relatively early period of life; in others, the bottom of the crevice remains on the enamel until late in life. This observation has led to a discussion as to whether or not the continued detachment of the epithelial attachment from the tooth surface and the final exposure of the cementum should be considered a pathological or a physiological process. Ever since Gottlieb's first publication on the epithelial attachment, there has been a diversity of opinion concerning this particular point, and at the present time the question is not settled. Gottlieb, Mueller, Orban, the author, and others consider the continued detachment of the epithelial attachment and the proliferation of the epithelium along the root surface a physiological process, as a part of the general aging of the organism. Another group of investigators, the best known of whom are Bauer, Becks, Häupl, and Lang, consider it a pathological process caused by mechanical injuries and subepithelial inflammation. The latter group of authors agrees that an epithelial attachment to the enamel exists when the tooth reaches the occlusal plane. But they consider only the condition present at that time as normal or physiological; any further detachment, and especially the proliferation of the epithelium along the cementum, is looked upon as a pathological manifestation.

Gottlieb speaks of the various successive stages illustrated in Figure 277 as "stages of eruption," the meaning of the word "eruption" being that the tooth under normal conditions continues to move occlusally throughout life, although at a much slower rate than in childhood. During this occlusal movement the gingival soft tissues are separated from the tooth at the bottom of the cervice. The crown is continuously worn off by abrasion; at the same time cementum is deposited on the surface of the root. So long as all these processes occur slowly and uniformly, they are expressions of a normal process of involution. By detachment of the gingival tissues at the bottom of the crevice, a fairly uniform length of clinical crown is maintained despite abrasion. By formation of cementum the root is

elongated enough to compensate for eruption, keeping the clinical root an adequate length.

There is no doubt but that the downward growth of the epithelium may also be the expression of a pathological process; this is especially true when it occurs abnormally fast, exposing large areas of the root at an early period of life. The pathological conditions resulting from deviations from the normal relationship between tooth and investing tissues will be discussed in Chapter XIII.

The rôle of subepithelial round-cell infiltration deserves special consideration since this infiltration is the main issue in the discussion of the proliferation of the epithelial attachment along the tooth surface. In most human specimens, inflammatory round cells (polyblasts) are found in the tissue bordering the crevice, and it does not seem very far-fetched to hold this inflammation responsible for the changes in the epithelium. However, it is the author's opinion that a direct correlation between inflammation and downward growth of the epithelium along the root surface cannot be demonstrated. Proliferation of the epithelial attachment along the cementum can be observed in specimens in which the bottom of the crevice and the subepithelial infiltration are distant from the epithelial attachment; on the other hand, specimens are found with severe inflammation but with no changes in the epithelium. The author is aware that it is impossible to settle this question definitely until human jaw specimens of individuals with excellent oral hygiene and healthy, normal gingivæ are available for histological examination. In such specimens the inflammatory factor could be excluded entirely, and it would then be possible to decide how the epithelial attachment acts in the absence of any inflammation. Up to this time, such specimens have not been reported.

EPITHELIAL ATTACHMENT AND GINGIVAL CREVICE IN ANIMALS.

The author, in collaboration with Ulrik, examined the teeth of small mammals that had not been subjected to the influences of domestication, among them the hedgehog (*Erinaceus europeus*). This animal fulfills all postulates necessary for a comparative investigation of the epithelial attachment in man and animals. It is omnivorous; its teeth resemble human teeth; and, like man, it wears down its teeth during life. All stages indicated in Figure 277 can be found in hedgehogs of different ages. In the young, the gingival tissues are united with the enamel by an epithelial attachment. The bottom of the gingival crevice is located on the enamel surface, and the

epithelial attachment ends at the cemento-enamel junction. Moreover, high cusps and thin cementum are found, analogous to the conditions in man. In older hedgehogs, the epithelial attachment grows apically along the surface of the cementum. In some teeth there is a slight subepithelial infiltration; in others there is no inflammation.

In old hedgehogs, the same regressive changes were observed as are found in man. The cusps are worn flat by occlusal wear; the size of the pulp chamber is greatly reduced by secondary dentin formation. Heavy layers of cementum are present on all root ends and in the bifurcation of the molars. The bottom of the gingival crevice has passed the cemento-enamel junction and is found on the root surface, so that the clinical crown includes part of the cementum surface. The depths of the gingival crevices in the old animals are about the same as in the young ones. The findings in these animals suggest that the identical changes in man are characteristic of all mammals with teeth similar to human teeth.

In this connection the forms of epithelial attachment in mammals with teeth structurally different from those of man will be considered. These animals can be divided into two major groups: rodents and herbivores.

The incisors of all rodents are continuously growing teeth: the loss of tooth substance by abrasion is replaced by an equal formation of new tooth structure at the apex. Unlike human teeth, they do not develop a fixed apical foramen. In the rat's incisors, the labial (outer) surface is covered with enamel over its entire length, and the lingual surface is covered with cementum. Consequently, the cemento-enamel junction is two lines running lengthwise on the mesial and distal surfaces of the tooth. Since the oral epithelium is attached to the entire circumference of the tooth at the bottom of the gingival crevice, there is an epithelial attachment to the enamel on the labial surface, and an epithelial attachment to the cementum on the lingual side. Like in man, the epithelial attachment migrates apically, so that a uniform length of clinical root and clinical crown is maintained.

The teeth of herbivores, such as the sheep, goat, and cattle, have long, enamel-covered crowns and relatively short roots. The outstanding characteristic of their teeth is that all of the enamel is covered with cementum, the enamel being exposed only where the occlusal surface has been worn. The epithelial attachment is always located on the cementum. It grows apically along the cementum surface and becomes detached from it at the bottom of the gingival crevice.

DEPTH OF THE GINGIVAL CREVICE.

The depth of the gingival crevice is subject to wide individual variation. Orban and Köhler measured 356 crevices on specimens of human teeth. Only crevices with an intact epithelial lining were measured. In 15 crevices the depth was zero. In 17 the depth varied from 0 to 0.1 mm. One hundred and twenty-nine crevices measured between 0.1 and 0.5 mm., 102 between 0.5 and 1 mm., 17 between 1 and 1.5 mm., 21 between 1.5 and 2 mm., 13 between 2 and 3 mm., and 2 between 4 and 6 mm. This gives an average depth of about 0.8 mm. However, these specimens were obtained from individuals who neglected oral care; in individuals with better oral conditions, shallower crevices would probably be found.

The age of the individual has but little influence upon the depth of the crevice; under good hygienic conditions, shallow crevices are found both clinically and microscopically in adults as well as in children.

The findings with regard to the depth of the gingival crevice can be summarized as follows:

The depth of the gingival crevice varies from zero to several millimeters; the majority of the crevices in human teeth measures between 0.1 and 1 mm.

The depth of the crevice is related to the health of the mouth; the cleaner the mouth, the shallower are the crevices.

The location of the crevice on the tooth surface has no influence upon its depth; shallow crevices may be found on the enamel surface in young individuals and on the cementum surface in older ones.

The shallower the crevice, the smaller are the chances for a possible development of pathological conditions in it.

Since the shallowest crevices have no depth at all, a depth of zero must be considered as the ideal, and therapeutic measures should attempt to create and maintain such crevices. This can be done by correct brushing of the teeth, careful oral prophylaxis, and avoidance of any kind of gingival irritation.

HISTOLOGY OF THE INTERDENTAL TISSUES.

All the specimens of human gingival crevices illustrated so far were taken from the labial or lingual side of the teeth. The conditions and changes that are found in the areas between the teeth will be considered next. Figure 278 shows the tissues between the crowns of the lower first and second bicuspid in a boy, aged eight years. Only about one-half of the crowns has erupted into the oral cavity.

The bottom of the gingival crevice is indicated by the presence of the enamel cuticle, *Cu*, and by the location of a slight round-cell infiltration. From *BC* to the cemento-enamel junction, *CEJ*, the epithelium is still in organic connection with the enamel. The gingival crevice measures about 0.05 mm., or practically zero.

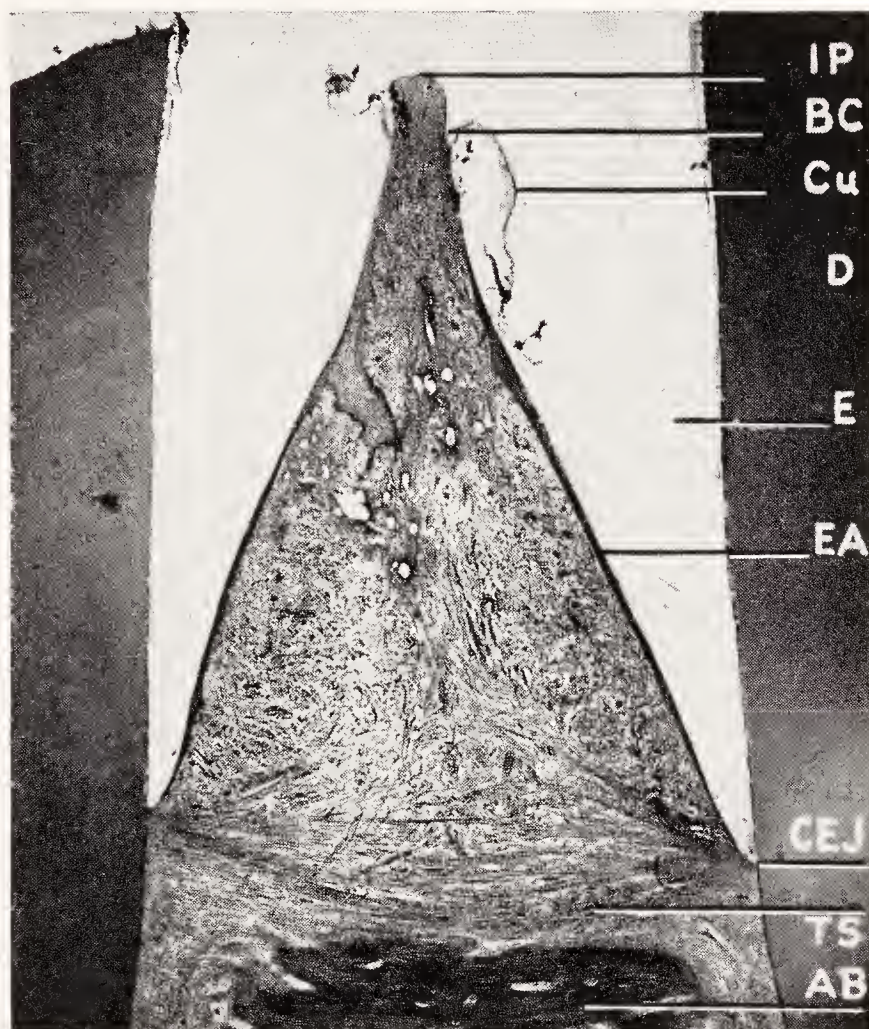


FIG. 278.—Interproximal space between the lower first and second bicuspid in a boy, aged eight years. The teeth have just erupted. The epithelial attachment extends from the cemento-enamel junction crownward almost to the level of the contact point. *IP*, tip of interdental papilla; *BC*, bottom of the gingival crevice; *Cu*, cuticle; *EA*, epithelial attachment to the enamel; *E*, enamel space; *D*, dentin; *CEJ*, cemento-enamel junction; *TS*, transeptal fibers; *AB*, alveolar bone.

A slightly different condition is found between the upper central and lateral incisors of a boy, aged fourteen years (Fig. 279). The bottom of the crevice is still located on the enamel surface; however, the width of the epithelial attachment on the enamel is much narrower than that found at eight years. The portion of the enamel between the bottom of the gingival crevice, *BC*, and the cemento-enamel junction is about one-fifth of the length of the anatomical crown. The epithelial attachment ends on both teeth at the cemento-enamel junction. The depth of the crevices is about 1.5 mm., measured from the tip of the interdental papilla to the bottom of the crevice at *BC*.

As the bottom of the crevice approaches the cemento-enamel junction, the epithelial attachment proliferates along the surface of the cementum. In the further process of detachment, the bottom of the crevice passes the cemento-enamel junction and is then found on the root surface. This condition is illustrated in Figures 280 and 281. In Figure 280 the bottom of the crevice is located on the cemen-

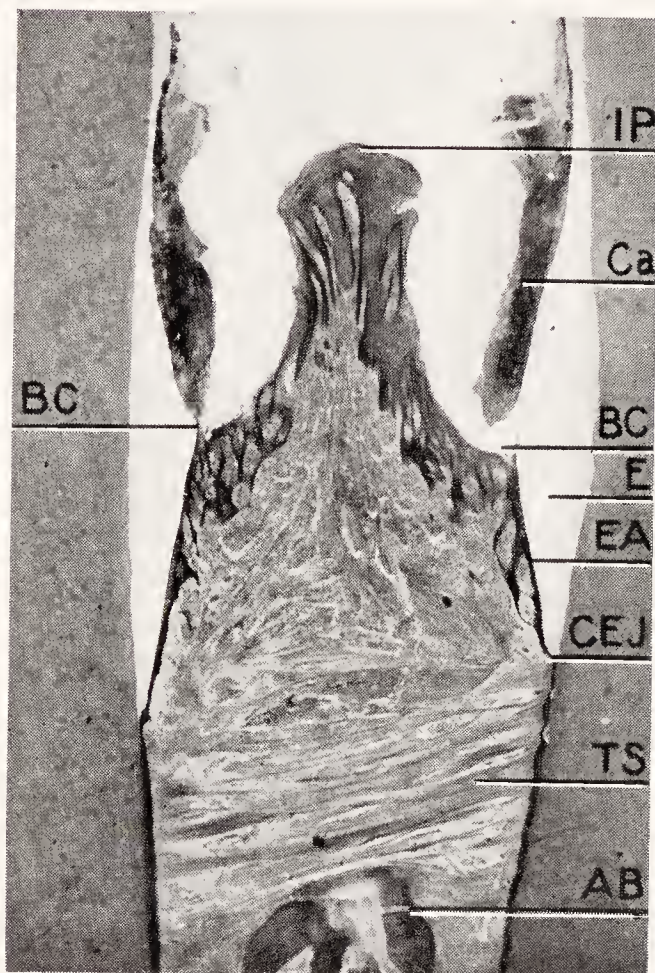


FIG. 279.—Interproximal space between upper central and lateral incisors of a boy, aged fourteen years. The bottom of the gingival crevice is located on the enamel; the epithelial attachment ends at the cemento-enamel junction. *IP*, tip of interdental papilla; *BC*, bottom of the gingival crevice; *Ca*, calculus in the gingival crevice; *EA*, epithelial attachment to the enamel; *E*, enamel space; *CEJ*, cemento-enamel junction; *TS*, transeptal fibers; *AB*, alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

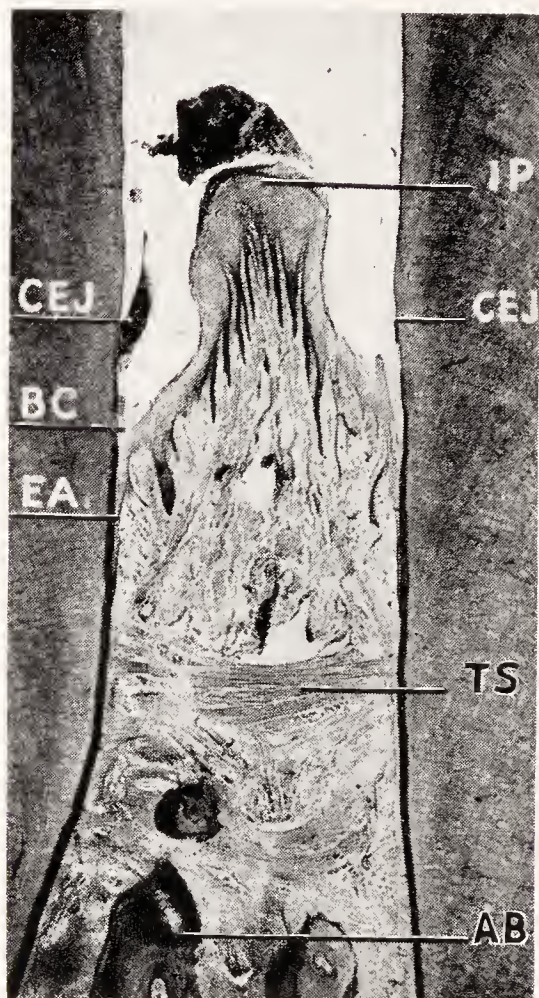


FIG. 280.—Interproximal space between two upper incisors in an adult. The bottom of the crevice has just passed the cemento-enamel junction and is on the root surface. *IP*, tip of interdental papilla; *BC*, bottom of gingival crevice; *EA*, epithelial attachment on the cementum; *CEJ*, cemento-enamel junction; *TS*, transeptal fibers; *AB*, alveolar bone.

tum, just beyond the cemento-enamel junction; in Figure 281 a considerable portion of the root surface is exposed between the cemento-enamel junction and the bottom of the crevice, *BC*. In both of these specimens the epithelial covering of the papilla is intact and shows a well-developed keratinized layer.

The transeptal fibers, which in Figures 278 and 279 are directly

below the cemento-enamel junction, are found further apically in Figures 280 and 281. The interdental alveolar bone is gradually shortened by resorption.

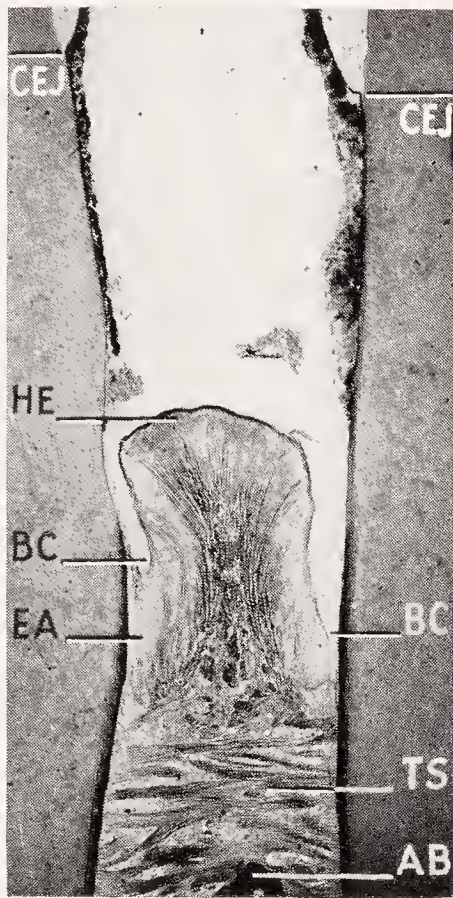


FIG. 281.—Interproximal space between two upper incisors in an adult. The bottom of the gingival crevice is located about 4 mm. rootward from the cemento-enamel junction. *HE*, hornified epithelium covering the interdental papilla; *BC*, bottom of the gingival crevice; *EA*, epithelial attachment on the cementum; *CEJ*, cemento-enamel junction; *TS*, transseptal fibers; *AB*, alveolar bone.

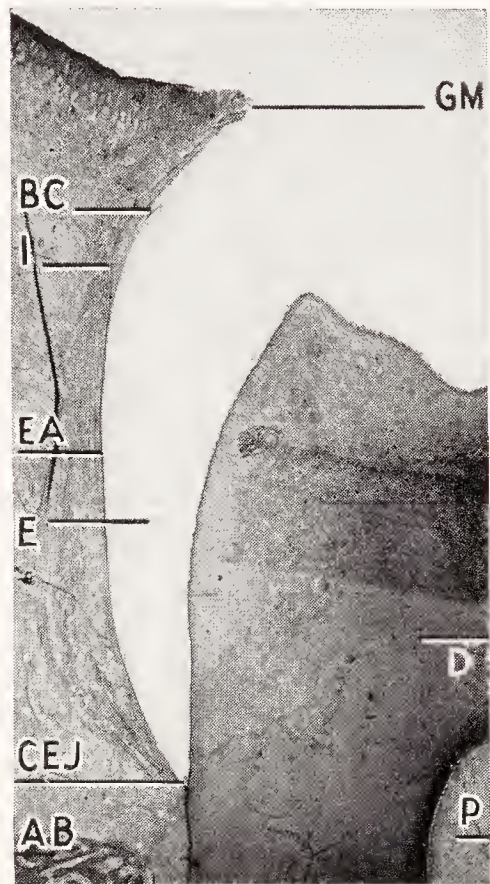


FIG. 282.—Distal surface of the crown of a lower first molar in a child, aged eight years. The distal cusps had just erupted. *GM*, gingival margin; *BC*, bottom of gingival crevice; *I*, subepithelial infiltration; *EA*, epithelial attachment to the enamel; *E*, enamel space; *CEJ*, cemento-enamel junction; *AB*, alveolar bone; *D*, dentin; *P*, pulp.

ANOMALIES OF THE EPITHELIAL ATTACHMENT AT THE CEMENTO-ENAMEL JUNCTION.

In the teeth of young individuals, the epithelial attachment to the enamel frequently does not extend all the way to the cemento-enamel junction but ends on the enamel surface. Thus, a small area of enamel next to the cemento-enamel junction is not covered with epithelium but is in contact with the surrounding connective tissue. This condition, which was first observed by Meyer, is illustrated in Figure 282, in the lower first molar of a child, aged eight years. The molar had just erupted; only about one-fourth of the length of the crown was visible in the mouth. The illustration

shows the epithelial attachment extending over almost three-fourths of the distal side of the enamel. The bottom of the gingival crevice (*BC*) is indicated by the thickening of the epithelium, the presence of the cuticle, and the location of the subepithelial infiltration. Toward the cemento-enamel junction, the epithelial attachment ends on the enamel surface (Fig. 283). If such a condition as this persists for a sufficient length of time, cementum may be deposited upon the enamel in this region, causing an overlapping of the cementum on the enamel (Fig. 284). Such extensions of the cementum over the enamel are called cementum spurs.

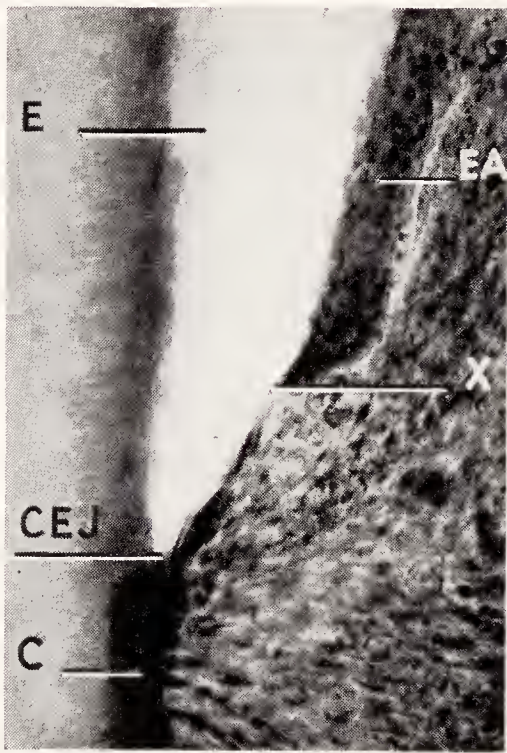


FIG. 283.—High magnification of cemento-enamel junction in Figure 282. *E*, enamel space; *C*, cementum; *CEJ*, cemento-enamel junction; *EA*, epithelial attachment to the enamel; *X*, deepest point of the epithelial attachment to the enamel. From *X* to *CEJ*, the enamel is in contact with the connective tissue.

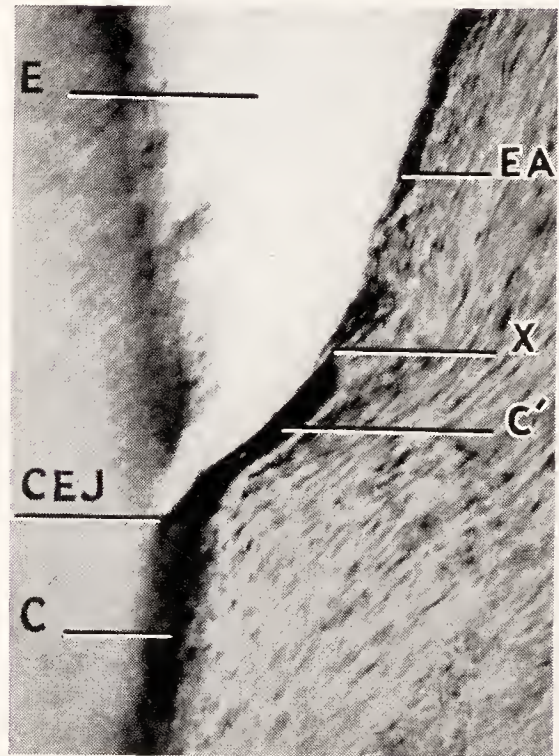


FIG. 284.—Deposition of cementum upon the enamel next to the cemento-enamel junction. *E*, enamel space; *C*, cementum; *CEJ*, cemento-enamel junction; *EA*, epithelial attachment; *X*, deepest point of the epithelial attachment to the enamel; *C'*, cementum deposited upon the enamel surface (cementum spur).

Cementum deposits on the enamel surface and spurs of cementum at the cemento-enamel junction are frequently found in embedded teeth. In such teeth the conditions leading to cementum deposition upon the enamel are the same as in Figures 283 and 284: loss of epithelial covering of the enamel, contact between enamel and connective tissue, and subsequent cementum deposition upon the enamel.

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CHAPTER XIII.

DISEASES OF THE PERIODONTAL TISSUES (GINGIVITIS, PYORRHEA).

NOMENCLATURE AND CLINICAL CONSIDERATIONS.¹

FEW subjects in dental pathology have caused more discussion and more diversity of opinion than so-called "pyorrhea alveolaris." This confusion is partly the cause and partly the result of the numerous attempts to introduce new names and terms and to create new clinical and pathological classifications. Pyorrhea alveolaris, paradentitis, periodontoclasia, and paradentosis are some of the more common terms; variations of these are paradontitis, parodontoclasia, and paradontosis. There seems to be hardly any reason for adding new terms to this existing variety.

It is the author's intention simply to describe and illustrate the various forms of periodontal diseases as they are found by the clinician and as they appear under the microscope. The author is convinced that any nomenclature of pyorrhea is useless unless there is a clear understanding of the pathological tissue changes associated with the various clinical forms. Lack of understanding of the basic principles of periodontal diseases makes it difficult to discuss intelligently symptoms and therapy, because identical terms are applied at random by different authors to etiologically widely different conditions.

The clinical terms recommended by Gottlieb will be used in this chapter. However, this does not mean that other terms are not as acceptable and suitable, provided they designate well-defined clinical and pathological pictures and thus can serve as a means of communication for diagnosis and therapy.

Gottlieb's clinical classification of periodontal diseases draws a definite line between the conditions developing as the result of

¹ At the Eighth International Dental Congress in Paris, 1931, the Committee on Terminology of the International Dental Federation recommended a nomenclature for periodontal diseases (see Transactions of the Eighth Dental Congress, Section IV). In the table on pages 316 and 317 these terms are put in brackets with the letters, F.D.I. Other terminologies, which are widely used in some parts of the United States and Canada, have been worked out by Box and by Becks. They are included in the table on pages 316 and 317, and are placed in the column to which they most closely correspond.

gingival inflammation and those developing as the result of systemic disturbances. In the following table, the author follows Gottlieb's classification with minor modifications that may help in correlating the clinical picture and the pathological manifestations.

The most common type of periodontal disease (A in Table, page 316) is the result of neglect. Gottlieb called this condition "Schmutzpyorrhœ," which term may be translated "pyorrhea due to uncleanness." Because of the accumulation of irritating débris and calculus in the gingival crevices, the tissues respond with hyperemia and engorgement until, finally, ulcers develop in the epithelial covering through which a purulent exudate is discharged. Gradually the inflammation progresses toward the alveolar margin, and inflammatory bone resorption takes place at the alveolar crest next to the superficial inflammation (marginal atrophy); the greater portion of the alveolar bone and periodontal membrane, however, remains intact. Clinically the teeth are firm and remain so except in extreme cases of long standing.

Another type of periodontal disease has a definite systemic cause (C II in Table, page 317). Its early clinical symptoms are primary bone lesions, resorptions of root surface and bone, widening of the periodontal space, and loosening of the affected teeth. Gottlieb called this condition "diffuse atrophy of the alveolar bone" to stress the difference between these lesions, which may occur anywhere on the root surface, and marginal atrophy, resulting from marginal inflammation (gingivitis). The resorptive processes on the root surfaces and alveolar bone and the resultant destruction of the periodontal membrane cause drifting of the teeth, known as pathological wandering; it may occur in a vertical direction (extrusion) or in a horizontal one (drifting, diastema formation). In diffuse atrophy, pocket formation and suppuration are usually late and secondary symptoms. In pathological wandering, the pocket develops on the side of the root from which the tooth is moving.

The clinical course of diffuse atrophy varies. Sometimes loosening and drifting come to a standstill after a period of several months; the teeth become firm in their new position and remain firm unless another attack of loosening and wandering takes place. Again, the destruction of the bone may be progressive; one tooth after another is lost until most or all of them have been exfoliated. Secondary infection of the pockets and secondary occlusal overstress, produced by the extrusion of the affected teeth, hasten the loosening.

In the terminal stages of bone destruction, when the teeth have lost most of their attachment and are very loose, diffuse atrophy

CLASSIFICATION OF PERIODONTAL DISEASES (PYORRHEA).

Clinical manifestations.	Etiology.	Pathology.	Therapy.
<p>A—Gingivitis, Schmutzpyorrhoe (Gottlieb). Gingivitis marginalis suppurativa (F.D.I.). Simplex periodontitis (Box). Primary paradentitis (Becks).</p>	<p>Local irritation of any kind: In most cases, soft and hard deposits (calculus) on the teeth. Neglect of oral hygiene. Gingival caries. Lack of contact point. Poor dental work, such as overhanging fillings, ill-fitting crowns, and other dental restorations that irritate the gingival soft tissues. Malalignment of the teeth.</p>	<p>In early stages, increased amount of subepithelial round cell infiltration in the superficial gingival tissue s. In more advanced stages, formation of breaks in the epithelial covering (ulceration) and discharge of pus. Inflammatory infiltration of the deeper gingival tissues. Resorption of the bone of the alveolar crest (marginal atrophy); otherwise periodontal membrane intact and of uniform thickness.</p>	<p>Removal of etiological factors: Scaling. Proper use of tooth brush. Improvement of oral health is followed by decrease in the inflammation and by healing of the gingival tissues.</p>
<p>B—Paradental pyorrhoea (Gottlieb). Paradentitis profunda (suppurativa) (F.D.I.). Periodontal pocket (Box). Secondary paradentitis or secondary paradentosis (Becks). Chronic suppurative pericementitis (Black).</p>	<p>Accelerated detachment of the epithelial attachment from the root surface without corresponding atrophy and recession of the soft tissues. Causes: (a) local causes: Habitual lack of oral hygiene, especially in the interproximal areas. Mechanical deepening of pocket (toothpick injury, food pack, etc.). Excessive lateral stress. (b) Systemic causes (see C II, diffuse atrophy of the alveolar bone).</p>	<p>Rapid proliferation of the epithelial attachment and detachment of the periodontal tissues from one side of the root; otherwise periodontal membrane healthy. Wall of pocket formed by inflamed connective tissue with ulcerated epithelial surface facing the root. Inflammatory destruction of alveolar bone around the pocket.</p>	<p>Removal of etiological factors: Scaling. Treatment of pocket: removal of outer wall of pocket (gingival flap) by packing, curettage, surgical excision, or electrocoagulation.</p>

C—Alveolar atrophy. I. Alveolar atrophy as result of excessive occlusal trauma.	Resorption of the alveolar bone as result of excessive occlusal trauma, (such as a single tooth exposed to excessive lateral stress after loss of adjacent teeth). Loosening of the teeth. Sometimes formation of pocket on the side toward which tooth is tipped. (Paradental pyorrhea, see B.)	Mutilation of mouth by multiple extractions. Shifting and tipping of remaining teeth with resulting lateral stress. Dental restorations, such as fillings or crowns, that interfere with normal occlusion and cause excessive occlusal stress. Failure of the tissue to repair repeated, minor traumatic injuries to the periodontal membrane and alveolar bone.	Resorption of alveolar bone on the side towards which tooth is pressed. Failure to compensate for loss of bone by reparative new-formation. Tooth embedded in fibrous connective tissue with varying degree of (secondary) inflammation.	Unless bone destruction is too far advanced, relief from occlusal stress by grinding or by an artificial restoration may bring about regeneration of bone.
II. Diffuse atrophy of the alveolar bone (Gottlieb). Dystrophiadiffusa (F.D.I.). Complex periodontitis (Box). Genuine paradentosis (Becks).	Loosening, pathological wandering of the teeth (elongation, drifting, rotation), and formation of diastemas between the anterior teeth are the earliest symptoms. The radiograph shows irregular and extensive loss of alveolar bone. Pocket formation and suppuration appear in later stages as secondary symptoms. In pathological wandering, the pocket appears on the side from which the tooth is drifting. Many cases of diffuse atrophy are observed in clean mouths that have been well cared for; often the patient is immune to caries.	Systemic or metabolic disturbances, such as diabetes. In some cases, etiology is glandular or dietary insufficiency; in others, etiology is unknown. Complete physical examination sometimes reveals the etiological factor or factors involved.	Presence of areas of resorption on the root surface. Resorption of alveolar bone. Widening of periodontal space with transformation of fibrous periodontal membrane into loose vascular connective tissue. These changes may occur anywhere on the root surface. If the disease involves areas of the root surface near the alveolar margin, rapid proliferation of the epithelial attachment along the root surface with subsequent deep pockets takes place; secondary infection and suppuration from these pockets usually ensue.	(a) Systemic treatment: Improvement of general health. Improvement of nutrition. If glandular deficiency is suspected, replacement therapy. (b) Local treatment: Grinding of affected teeth so that they are out of occlusion. Balancing of occlusion. Prophylactic checking of overbite and lateral stress. Symptomatic treatment of pockets. Mechanical fixation in early and favorable cases.

can no longer be distinguished from pyorrhea due to calculus and gingivitis. However, since nothing can be done for such an advanced condition, the differential diagnosis is of no special importance.

Paradental pyorrhea (B in Table, page 316) is a connecting link between marginal inflammation and diffuse atrophy, since it may accompany either one of these conditions. Clinically, it can be defined as a condition in which discharge of pus from the pockets persists despite removal of all local irritation. In other words, the pockets are so deep that purulent inflammation is perpetuated despite scaling, brushing, or massaging. Such pockets may be caused by habitual lack of cleanliness and chronic inflammation, especially in the interproximal spaces, by mechanical injuries to the bottom of the gingival crevice, or by excessive lateral stress with resulting destruction of the periodontal attachment and pocket formation on the side toward which the tooth is pressed.

Sometimes, paradental pyorrhea is a symptom accompanying diffuse atrophy; the loss of bone and periodontal attachment is followed by rapid detachment of the soft tissues alongside the root and subsequent formation of a deep pocket in the affected area.

Nowadays most investigators agree that there are the two kinds of pyorrhea: marginal inflammatory conditions of local origin, and primary bone changes of systemic etiology with suppuration as a late symptom. Box's terms, simplex and complex periodontitis, and those of Becks, marginal paradentitis (primary paradentitis due to calculus) and genuine paradentosis (primary bone disturbance) obviously follow this division. Häupl and Lang differentiate between a superficial form of periodontal lesion (paradentitis marginalis superficialis) and a deep form (paradentitis marginalis profunda). Their clinical description corresponds to Gottlieb's Schmutzpyorrhœ and diffuse atrophy: paradentitis marginalis superficialis is characterized clinically by marginal gingivitis and firm teeth, paradentitis marginalis profunda by loosening and pathological wandering. Häupl and Lang, however, see in the deep lesions only the continuation of the superficial inflammation and do not recognize a systemic component or a primary deep bone lesion.

All investigators agree that the local form of pyorrhea is far more frequent than true diffuse atrophy. Among 500 patients Black found only 5 per cent entirely free of gingivitis; of the remaining 95 per cent, Black attributed about 40 per cent to calculus, 37 per cent to imperfect dental operations, and 18 per cent to malformations and malpositions of teeth. Gottlieb claimed that 90 per cent of all pyorrhea is due to neglect and uncleanness.

A few words must be said about the bacteriology of pyorrhea. Pyorrhea is not a specific infection. The various forms of microorganisms and protozoa (amœbæ) found in the pus of pyorrhea pockets are partly saprophytes and partly pathogenic microorganisms that grow in the depths of the pockets and in the inflamed tissues. However, these microorganisms have no etiological relationship to pyorrhea. In every instance of suppurating pockets, the pocket formation is the primary factor and the infection a secondary complication; as soon as the pocket is eliminated or the tooth extracted, the bacteria disappear promptly.

ALVEOLAR ATROPHY.

Alveolar atrophy is caused by resorption of the alveolar bone with insufficient bone regeneration. The causes for the resorptive process leading to bone atrophy can be classified as follows:

Physiological alveolar atrophy during the general involution with advancing age.

Alveolar atrophy due to inflammatory processes in the gingival tissues.

Alveolar atrophy due to excessive occlusal stress.

Alveolar atrophy as a symptom of a systemic disturbance (diffuse atrophy).

Physiological Atrophy of the Alveolar Margin With Advancing Age (Senile Atrophy).—In order to clarify the significance of the physiological alveolar atrophy that takes place throughout life, it is necessary to recall briefly the age changes that occur in every normal healthy tooth:

(a) Enamel: mechanical abrasion from mastication.

(b) Dentin: continual deposition and sclerosis of the dentin; formation of secondary dentin wherever the dentin surface has been exposed by abrasion.

(c) Pulp: decrease in the size of the pulp chamber; fibrosis and calcification of the pulp tissue and atrophy of the pulp cells and vessels.

(d) Cementum: continual deposition of cementum, resulting in a thickening of the cementum, especially at the apices of all teeth and in the bifurcation of multirooted teeth.

In the investing tissues the following involutionary changes occur:

(e) Epithelial attachment and gingival crevice: the deepest point of the epithelial attachment grows rootward. At the same time, the epithelial attachment is detached at the bottom of the

crevice. As a result, with advancing age, the bottom of the crevice passes the cemento-enamel junction, and is found on the root surface.

(f) Topographic relationship between tooth and socket: the teeth move occlusally throughout life. In youth, during the eruptive period, this movement is comparatively fast. Later in life it is much slower. In addition there is a slow mesial drift of all teeth. Both this occlusal movement and mesial drift occur at the rate at which tooth structure is lost at the occlusal surface and at the contact points by occlusal and interproximal wear, respectively.

(g) Alveolar bone: the amount of alveolar margin that is resorbed is determined by the atrophy of the gingiva and the extrusion of the tooth from its socket. This bone atrophy finally leads to what is known clinically as senile alveolar atrophy.

The alveolar atrophy described under (g) is part of the normal life cycle of every tooth. There is great variation in the rate at which it occurs; the slower the rate, the better is the prognosis for the teeth. Physiological alveolar atrophy is clinically recognized by the gradual shortening and rounding of the interdental bone crests, which can be noticed in radiographs of adults and old persons with firm teeth and healthy gingival tissues.

Under pathological conditions, alveolar atrophy may occur rapidly, thus producing in relatively young individuals a condition normally observed only in much more advanced age. The etiology of this premature atrophy of the alveolar process is unknown; the term "atrophia præcox" has been proposed for it.

Alveolar Atrophy Caused by Inflammatory Processes in the Gingival Tissues.—Any gingivitis that involves more than the superficial gingival tissues is likely to cause resorption of the bone at the alveolar crest. The mechanism of this bone resorption is the same as in every inflammatory process occurring in the neighborhood of bone: the inflammation causes hyperemia of the soft tissues and resorption of the adjacent bone. There is no true osteitis or osteomyelitis; the common reference to "necrotic bone" in pyorrhea is based upon a misconception. The bone is resorbed by osteoclasts, but it is not exposed and does not become necrotic, as, for instance, in osteomyelitis of the jaws.

Inflammatory bone resorption in gingivitis is confined to the alveolar crest; the deeper bone and periodontal membrane are intact. Clinically this is indicated by the firmness of the affected teeth. Inflammation of long standing may eventually reduce the height of the bone to such a degree that the tooth becomes loose.

However, it usually takes decades of extensive marginal inflammation to loosen a tooth in that way.

The prognosis of marginal atrophy is decidedly favorable in the early stages. If the inflammation subsides, bone resorption comes to a standstill, and although the periodontal attachment does not regenerate in the areas in which the bone has been destroyed, the remaining portion of it is strong enough to insure retention of the tooth.

Alveolar Atrophy Due to Excessive Occlusal Stress.—Excessive occlusal stress, particularly in a lateral direction, may cause resorption of the alveolar bone on the side of pressure. If the force exerted upon the tooth is not too great or of too long duration, the loss of bone can be compensated for by deposition of new bone and cementum, and the attachment of the tooth on the side of pressure is regenerated. If, however, the force is excessive, more and more bone may be resorbed until the periodontal space becomes very wide; the fibrous periodontal membrane is replaced by loose connective tissue, and the tooth is loosened (see Fig. 308).

The prognosis of alveolar atrophy resulting from excessive occlusal stress depends upon the extent of bone destruction and the reparative power of the body. If the loosening of the tooth is not yet excessive, relief from the trauma will frequently be sufficient to start bone regeneration. If the bone atrophy is advanced, the tooth is usually lost.

Alveolar Atrophy as a Symptom of a Systemic Disturbance.—Extensive alveolar atrophy occasionally occurs without a local cause. It must then be considered as the oral manifestation of some, often unknown, systemic disturbance. A group of radiographs that illustrates the rapidity with which alveolar bone may be destroyed as a result of systemic disturbances is reproduced in Figures 285 and 286. The patient, a man, aged forty-four years, suffered from diabetes mellitus. During the twenty months of observation, the alveolar bone atrophied extensively; in some teeth, such as the upper left cuspid, the upper right third molar, and the lower central incisors, there is practically no bone left, although less than two years before these teeth were firmly supported by bone.

Loosening of the teeth, loss of supporting bone, rapid pocket formation, and development of lateral abscesses are symptoms of diabetes which sometimes may lead to an early diagnosis of the disease (Hirschfeld). Several diabetic patients have been observed in whom the oral manifestations improved greatly after proper medical treatment of the disease had been started.

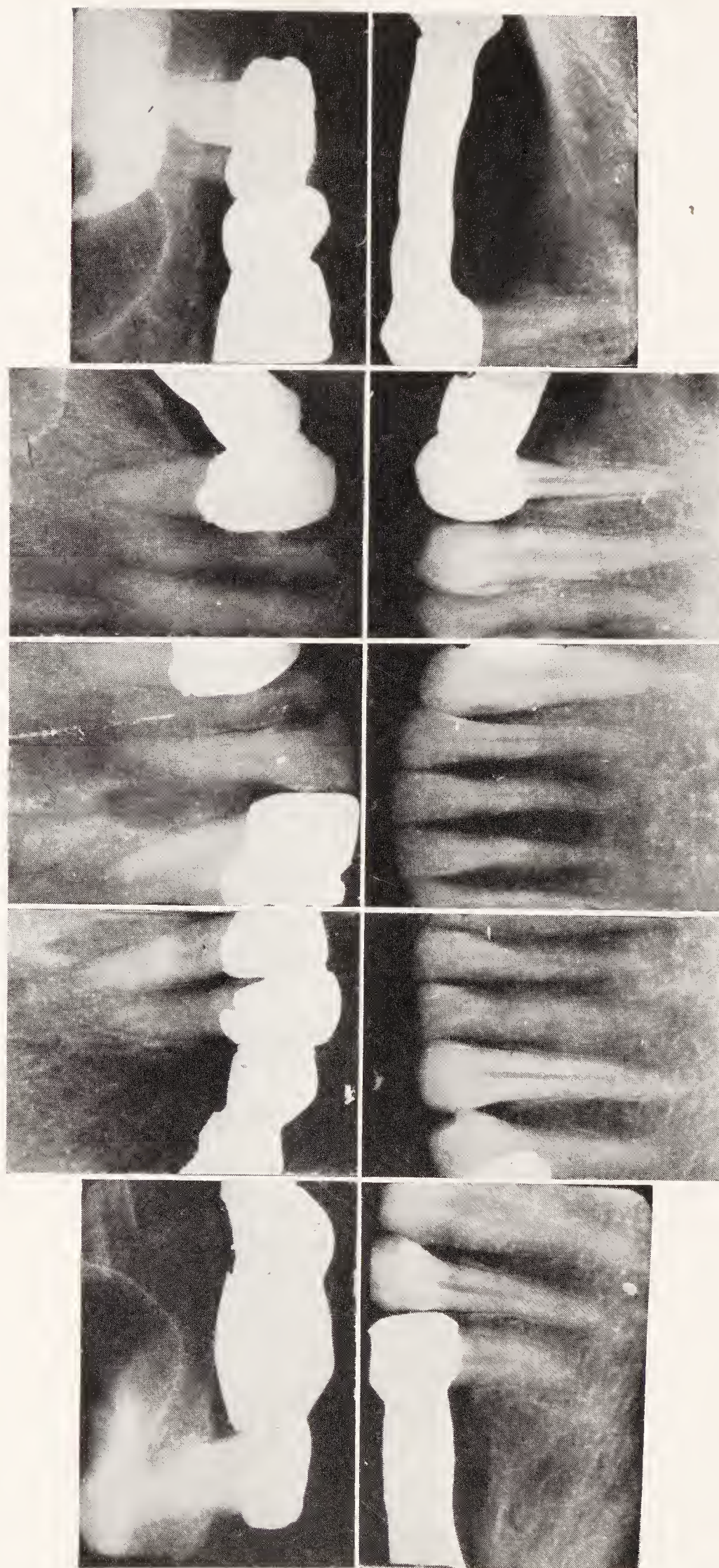


FIG. 285

FIGS. 285 and 286.—Radiographs of a man, forty-four years old, suffering from diabetes mellitus. The radiographs in Figure 285 were taken on April 14, 1934.

Figure 286 (opposite page), December 13, 1935. A comparison of the radiographs of the two periods reveals the amount of bone destruction that occurred during the interval of twenty months. (Courtesy of Albert A. Dahlberg.)

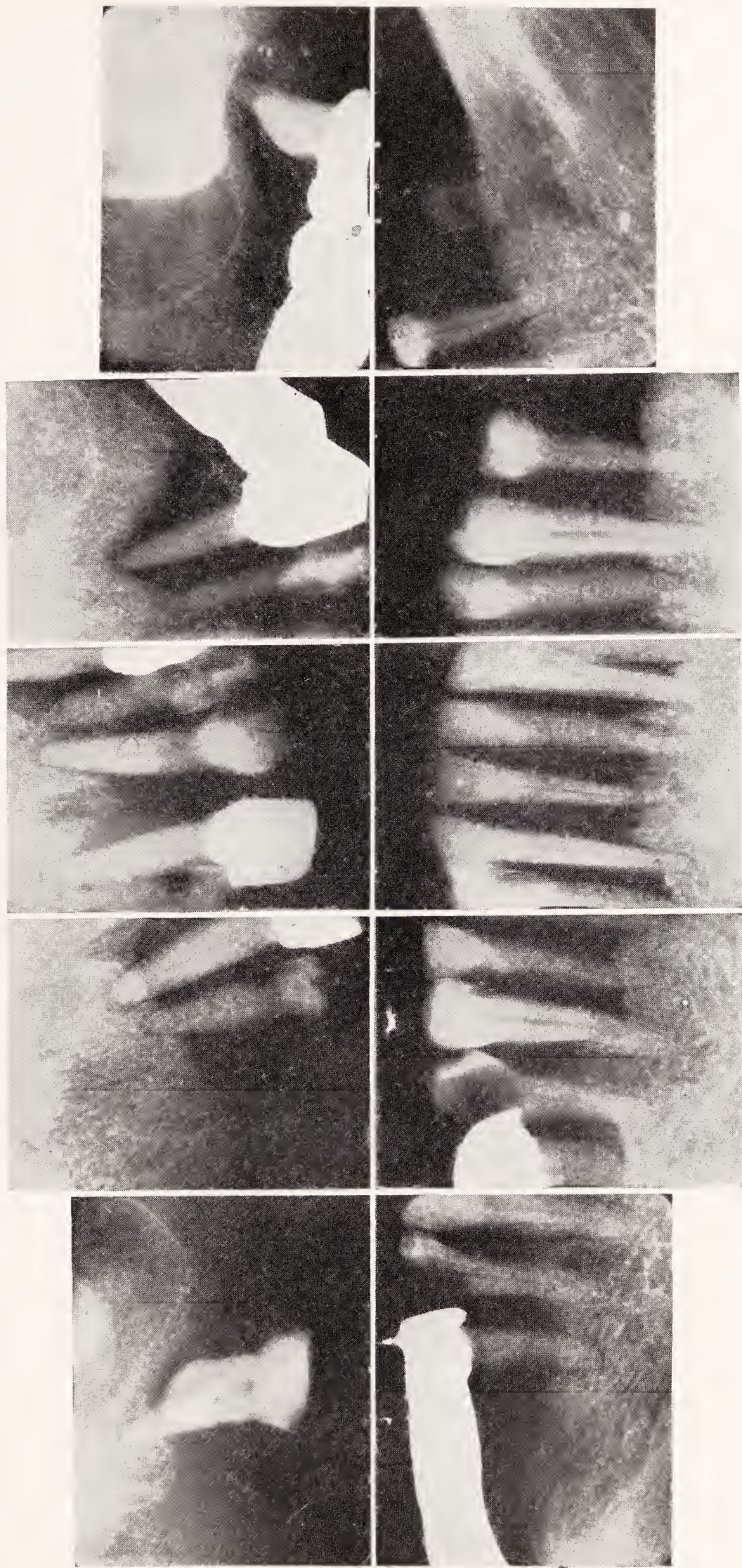


FIG. 286

In some cases of primary bone atrophy only one or a few teeth in the whole mouth are affected. Sometimes an anterior tooth becomes loose, elongates, and drifts (pathological wandering); the change in the position is often first noticed clinically by the formation of an increasing space between the affected tooth and its neighbors. If a posterior tooth drifts, it almost invariably assumes a position in which it is subjected to excessive occlusal stress, which, in turn, hastens its loosening.

Frequently diffuse atrophy is observed in the mouths of individuals with large, well-built teeth, little calculus, and immunity to dental caries.

The etiology of diffuse atrophy is not yet known. Metabolic disturbances, faulty nutrition, endocrine dysfunction, pregnancy, and diabetes are some of the conditions sometimes associated with it. So far as diet in relation to diffuse atrophy is concerned, the author wishes to caution against hasty conclusions. Most of the lesions produced experimentally in the teeth and alveolar bone of animals by dietary measures bear only a remote resemblance to diffuse atrophy in man. The food of these animals is unlike any human diet; besides, in animals the pathological bone changes were not confined to the jaw bone but were found throughout the skeleton, whereas in most patients suffering from diffuse atrophy the skeleton is radiographically normal. Therefore, it may be better to admit ignorance of the etiology of diffuse atrophy until more information is available.

HISTOPATHOLOGY OF GINGIVITIS DUE TO LOCAL IRRITATION.

Clinical Considerations Concerning Calculus.—Calculus, the main source of gingival irritation, is the result of the calcification of organic matter deposited upon the clinical crowns of teeth. This deposit consists of desquamated epithelial cells of the oral mucosa, food débris, salivary mucin, and bacteria. The first step in the formation of calculus is the deposition of these substances, which at first can easily be wiped off during mastication or with a tooth-brush; calcification and hardening of this film by the deposition of calcium salts from the saliva are secondary.

The chemical composition of calculus is variable. The latest analyses are those reported by Glock and Murray, who give the following average figures for the composition of salivary calculus:

CALCULATED COMPOSITION OF SALIVARY CALCULUS.

Inorganic:

CaCO ₃	3.17 per cent
Ca ₃ (PO ₄) ₂	75.97 per cent
Mg ₃ (PO ₄) ₂	3.77 per cent
	<hr/>
	82.91 per cent
Protein	8.34 per cent
Fat	2.71 per cent
Water, etc.	6.04 per cent

The protein was found to consist of keratin, mucin, and nucleoprotein. Hard calculus contains a slightly higher percentage of inorganic salts, soft calculus a slightly higher percentage of organic matter and water.

Calculus formation varies greatly with different individuals. Some mouths are practically free of it; in others it forms rapidly despite good oral care and frequent scaling. The reasons for this individual difference are not known.

Calculus is most commonly deposited on the lingual surfaces of the lower anterior teeth, opposite the openings of the submaxillary and sublingual glands, and on the buccal surfaces of the upper posterior teeth, opposite the opening of the parotid duct. But it may be found wherever there is a possibility of undisturbed accumulation of organic matrix and subsequent incrustation with calcium salts.

For clinical description, calculus is divided into supragingival or salivary calculus and subgingival or serumal calculus. There is no difference in the etiology of these two types: both originate from the saliva, and develop by an incrustation of soft, organic deposits with salivary calcium salts. Salivary or supragingival calculus is found mainly near the openings of the salivary glands, namely, on the lingual surface of the lower anterior teeth and on the buccal surface of the upper molars. Usually it has a light yellow color, unless smoking has turned it brown. Since it is attached to the tooth surface crownward from the gingival margin (see Fig. 291), it is easily visible. Serumal or subgingival calculus, on the other hand, may be found on any tooth in the mouth; it has a dark brown or greenish color and is hard and brittle. It is deposited on the tooth surface located inside of the gingival crevice, between the gingival margin and the bottom of the crevice; therefore, it is partly or entirely hidden and can be detected only with an explorer.

The difference in the location of salivary and serumal calculus is probably the cause for the difference in their physical qualities.

Salivary calculus, being deposited near the sources of saliva, forms rather rapidly. Because of its accessible location, it is constantly bathed in saliva; thus, all pigmentations that may originate from various causes are washed out, and the calculus retains its light color. Serumal calculus, on the other hand, forms more slowly; under the protective cover of the gingival crevice, it becomes denser and harder than salivary calculus. Small hemorrhages from the ulcerated soft tissues add blood pigment to it, which gives it a dark color.

Calculus can be deposited only on the clinical crown, that is, on the tooth surface that is crownward from the bottom of the gingival crevice and that is accessible to oral fluids. The bottom of the gingival crevice is the limit of deposition of any kind.

Gingivitis Due to Lack of Oral Care and to Calculus.—In the microscopic study of specimens of human gingivæ, it is difficult to draw a line between what is normal and what is an early pathological manifestation. As has been pointed out in the discussion of the epithelial attachment and gingival crevices, microscopic research along this line is considerably handicapped by the restrictions concerning autopsy material. Many of the individuals from whom jaws are obtained during autopsy had almost no oral care and died after a long and severe illness in some public hospital. Consequently, there are soft and hard deposits and gingivitis in most human jaw specimens.

Although, as a rule, the mouths of children are comparatively clean and free from deposits, the early stages of marginal gingivitis and calculus formation can frequently be observed in early ages. Since in young teeth the bottom of the gingival crevice is located on the enamel surface, calculus is deposited on the enamel, and the resulting gingivitis is found in that portion of the gingivæ that is attached to or overlies the enamel.

Figures 287 and 288 show the labial side of a lower cuspid and the lingual side of a lower central incisor of a boy, aged fourteen years. The mouth was in good condition; no teeth were missing, and only small occlusal carious lesions were present in the first molars. The general appearance of the specimen, which showed a considerable amount of soft deposits on some of the teeth, suggested that during the last few weeks of life oral care had been neglected. The condition of the gingival tissues varied according to the degree to which the various tooth surfaces were self-cleansing. Hence the difference between the microscopic appearance of Figures 287 and 288. On the labial surface of the lower cuspid, a habitually rather clean area, the hornification of the gingival epithelium extends to the bottom

of the shallow crevice. Only a small amount of subepithelial round-cell infiltration is present. The epithelium is still attached to the gingival one-fourth of the enamel. As a whole, this condition can be considered normal and typical for the particular age and stage of eruption.

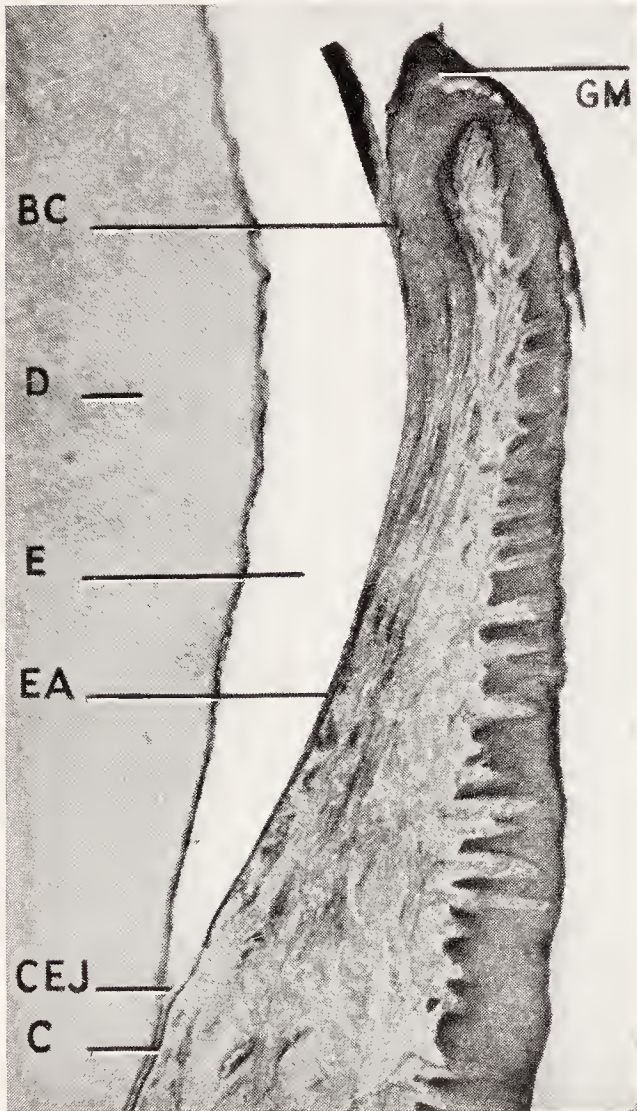


FIG. 287.—Normal gingival crevice on the labial surface of a lower cuspid of a boy, aged fourteen years. The depth of the crevice is practically zero. The hornification of the gingival epithelium extends to the bottom of the crevice. Very small amount of subepithelial infiltration. *D*, dentin; *E*, enamel; *BC*, bottom of gingival crevice; *EA*, epithelial attachment to the enamel; *CEJ*, cemento-enamel junction; *C*, cementum; *GM*, hornification of the epithelium at the gingival margin.

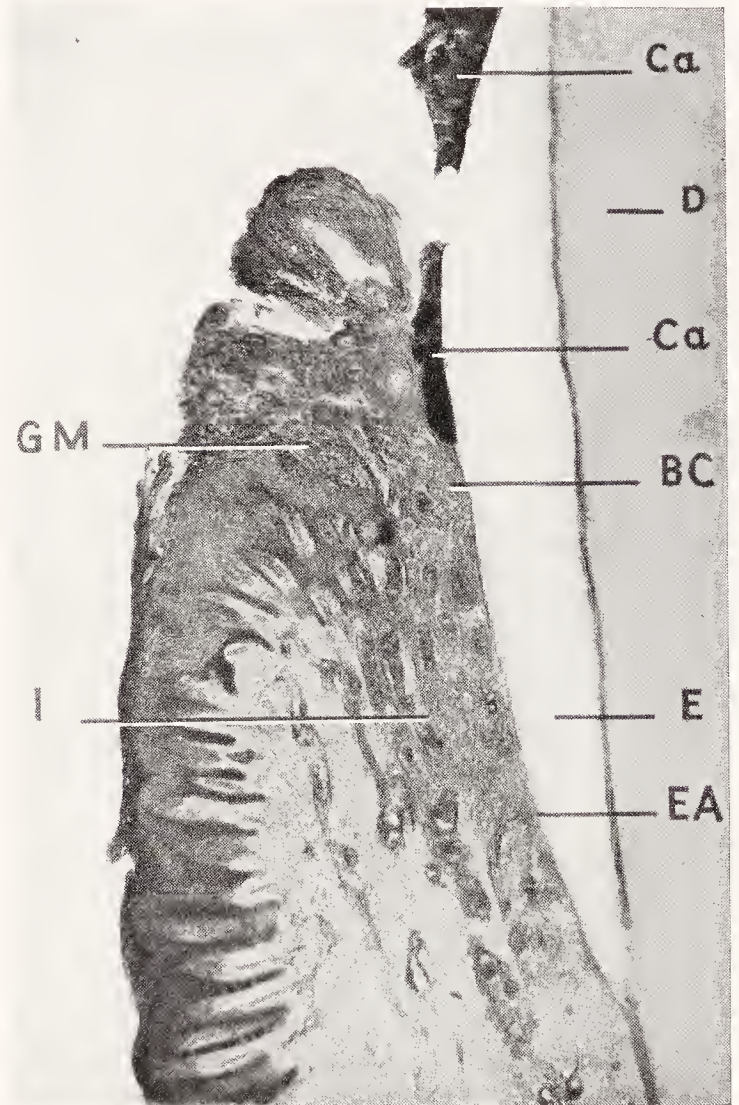


FIG. 288.—Early stage of gingivitis due to lack of oral care. Lingual surface of lower central incisor of the same individual from which Fig. 287 was taken. *D*, dentin; *E*, enamel; *BC*, bottom of gingival crevice; *EA*, epithelial attachment to the enamel; *Ca*, calculus on the enamel surface; *GM*, ulcerated gingival margin covered by masses of desquamated epithelial cells and by soft deposits; *I*, subepithelial round-cell infiltration and capillary hyperemia.

The presence of a small number of round cells in the subepithelial tissues can be considered an indication of gingivitis; but if it is, almost every human gingival crevice would have to be considered pathological, which would only cause confusion. Therefore, the author will follow the practice of calling gingivæ and gingival crevices with intact, well-hornified epithelium, such as the one illus-

trated in Figure 287, normal, despite the presence of a small amount of subepithelial infiltration; the terms "gingivitis" and "pathological crevices" will be used whenever the specimens show distinct breaks and defects in the epithelial covering and a marked inflammatory reaction in the subepithelial connective tissue.

As stated before, the location of the crevice on the tooth surface has no significance whatsoever in deciding whether a crevice is normal or pathological. The bottom of a crevice may be located on the surface of the enamel, and yet the condition may be distinctly pathological if the crevice epithelium is ulcerated and the subepithelial tissue is extensively inflamed; on the other hand, in an older person, the bottom of a crevice may be on the cementum surface, but the intact epithelial covering, lack of inflammatory symptoms, and clinically healthy appearance justify calling such a crevice normal.

The lingual surface of the lower central incisor of the same individual, aged fourteen years, shows symptoms of the beginning of gingivitis (Fig. 288). The crest of the gingiva is covered with desquamated epithelial cells. The crevice epithelium and subepithelial tissue show marked inflammatory changes. Dense round-cell infiltration extends to the cemento-enamel junction. The capillaries are enlarged and hyperemic. At the bottom of the crevice the epithelium has been lost, and inflammatory exudate cells are being discharged into the crevice. The alveolar bone is not affected; the inflammatory changes are still confined to the gingival margin and have not yet involved the deeper tissues.

In specimens of adults with calculus and inflammation, the bottom of the gingival crevice, as a rule, is found on the surface of the root. In such teeth it is hard to decide whether the inflammation was superimposed upon the recession, or whether it caused the recession, or whether both inflammation and recession existed independently. Skillen made a comparative study of depths of the pocket, inflammation, and epithelial proliferation and came to the conclusion that "It is impossible, or at least difficult, to correlate the conditions in and about the gingiva with the epithelial proliferation, although these conditions seem to play some rôle in the so-called recession of the gingiva."

Figures 289 and 290 show the lingual side of an upper cuspid in an adult. The bottom of the crevice is located slightly rootward from the cemento-enamel junction. A piece of calculus lies in the pocket, extending into the pocket epithelium and causing a break in the epithelial continuity between gingiva and epithelial attach-

ment. The subepithelial connective tissue shows dense inflammatory round-cell infiltration which extends along the root surface apically until close to the alveolar crest. There is evidence of resorption in the latter, no doubt as a result of the approaching gingival inflammation. The crevice is very shallow.



FIG. 289

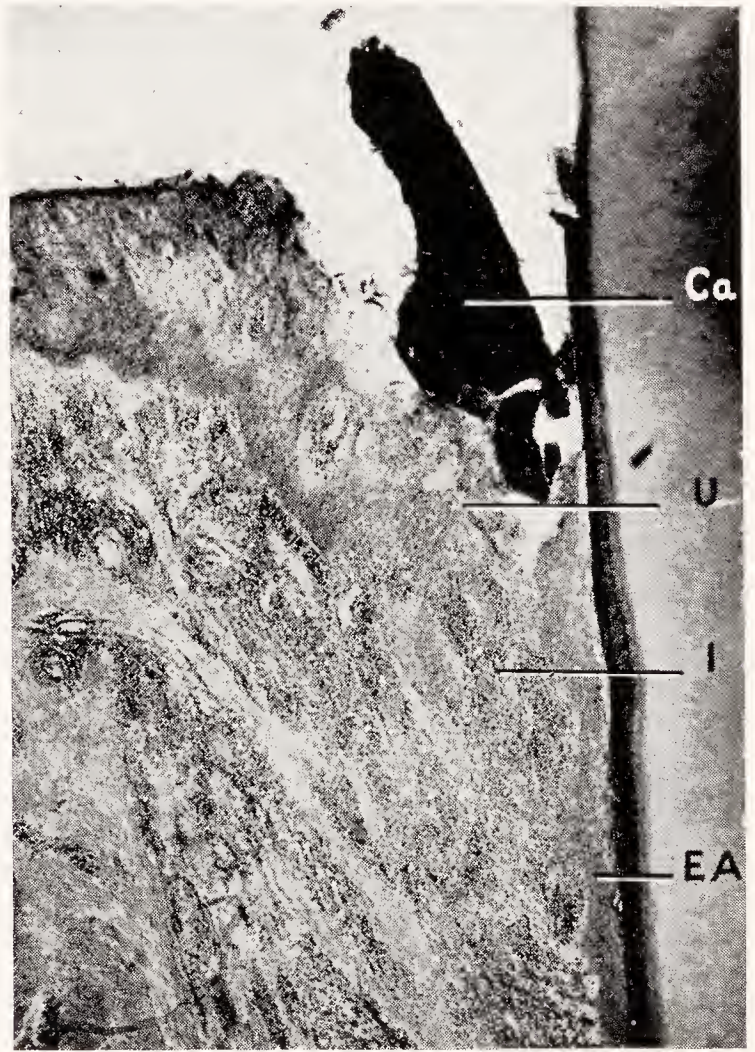


FIG. 290

FIGS. 289 and 290.—The effect of calculus upon the gingival tissues. Ulceration of the gingiva. Lingual surface of an upper cuspid in an adult.

FIG. 289.—*D*, dentin; *CEJ*, cemento-enamel junction; *Ca*, calculus at the bottom of the crevice; *I*, dense inflammatory infiltration of the tissues beneath the calculus; *GE*, gingival epithelium; *EA*, epithelial attachment to the cementum; *R*, resorption of the alveolar crest; *PM*, intact periodontal membrane.

FIG. 290.—Higher magnification of Figure 289. *Ca*, calculus; *U*, ulceration below the calcific deposit; *I*, inflammatory exudate cells; *EA*, epithelial attachment to the cementum.

As can be seen in subsequent illustrations, the relationship between marginal inflammation, calculus, and depth of pocket varies widely. Apparently some individuals tend to develop a productive or proliferative inflammation with tissue formation, while others are more likely to develop an ulcerative form of inflammation with tissue destruction. In some cases of chronic gingivitis, the pockets are deep; in others they remain shallow. In the first case, the free gingivæ have become hypertrophic or at least they have failed to

shrink. The bottom of the crevice moved apically, and the distance between it and the gingival margin became greater, causing a deepening of the pocket. In the second case, inflammation and ulceration destroyed the gingival margin, and although the detachment of the tissues from the tooth surface progressed apically, the pocket did not become deeper.

The prognosis for either form of marginal gingivitis is favorable; the soft tissues readily respond to removal of the irritation and



FIG. 291.—Deposits of salivary calculus on the buccal surface of an upper molar. The calculus lies flat upon the ulcerated surface of the gingiva. *Ca*, calculus; *U*, ulcer; *I*, inflammatory infiltration; *GE*, gingival epithelium; *AB*, alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

proper care. As a rule, healing is more rapid if the pockets are shallow than if they are deep. The healing of the gingival tissues can be understood from Figure 290, which shows a fragment of calculus in the gingival tissues at the bottom of the crevice. To the left of the calculus, the oral mucosa is intact; to the right, the epithelial attachment covers the cementum surface. Directly beneath the calculus an ulcer has developed; the subepithelial tissue shows extensive inflammatory infiltration with round cells, some of which have passed through breaks in the epithelium and are discharged from beneath the calculus. If the calculus were removed and the gingivæ were kept clean by proper brushing and care, the subepithelial inflammation would disappear within a few days;

at the same time, epithelium would begin to proliferate from the mucosa of the gingiva as well as from the epithelial attachment until the entire surface of the former ulcer would be covered with a new intact epithelial layer.

A large amount of calculus may accumulate and the gingival tissue may be extensively destroyed without the formation of a pocket. In such a case the calculus is located on top of a flat,

ulcerated gingival surface; pus is discharged from the space between the base of the deposit and the underlying gingival tissue. Figure 291 illustrates a condition of this type. The specimen shows the buccal side of an upper second molar the crown of which was covered with a flat, chalky, yellow deposit of salivary calculus whose thickness increased toward the gingiva. On the surface of the underlying tissue is a flat ulcer; the subepithelial connective tissue is densely infiltrated and inflamed, and the buccal plate of the

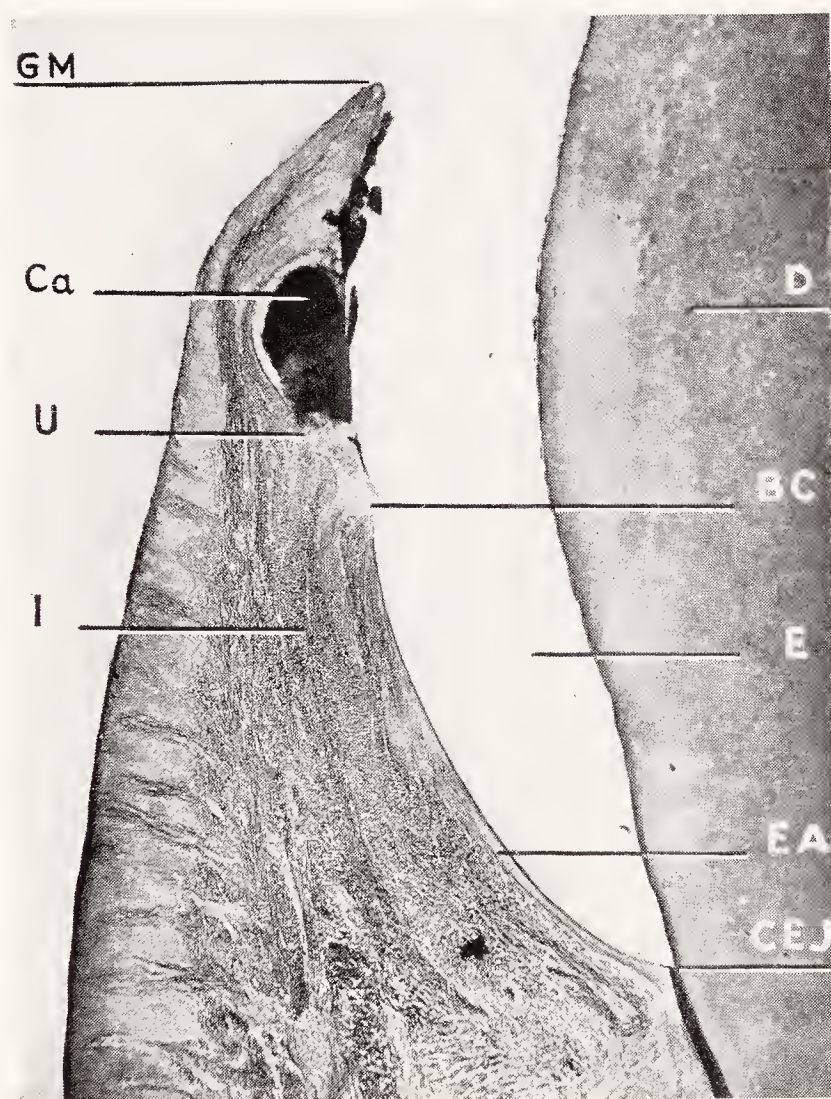


FIG. 292.—Subgingival calculus on the surface of the enamel. Lingual surface of a lower deciduous molar. *D*, dentin; *E*, enamel; *BC*, bottom of gingival crevice; *Ca*, calculus; *U*, ulceration of the wall of the crevice overlying the deposit; *GM*, gingival margin; *I*, subepithelial infiltration; *EA*, epithelial attachment to the enamel; *CEJ*, cemento-enamel junction.

alveolar bone has an uneven, resorbed surface. There is an epithelial attachment to the surface of the cementum. After removal of the calculus, this epithelium, the epithelium at the gingival margin, and the islands of epithelial tissue within the ulcer are sources from which a new epithelial covering develops and within a few days covers the gingival tissue, thus reestablishing normal conditions.

Up to this point all specimens that have been shown have had shallow crevices and supragingival calculus. The following group

of specimens illustrates the histopathology of the gingival tissues in the presence of subgingival calculus in pockets of considerable depth. If subgingival calculus develops in childhood, it is found on the surface of the enamel. Figure 292 shows such a case taken from the lingual side of a second deciduous molar of a child, aged eight years. The bottom of the crevice is located on the enamel; between it and the gingival margin, subgingival or serumal calculus

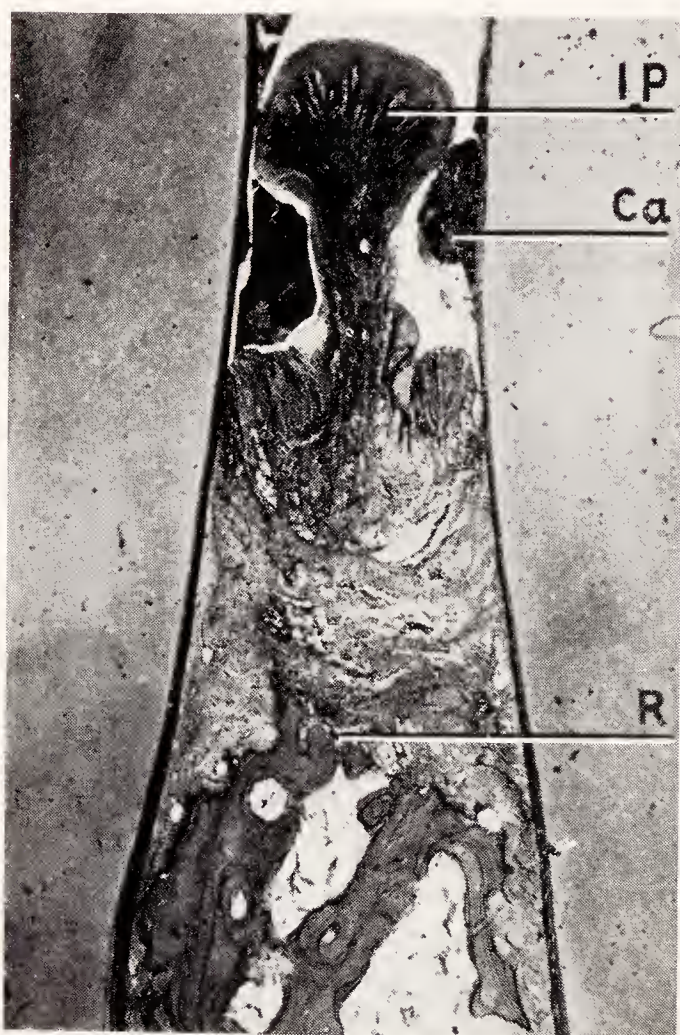


FIG. 293.—Subgingival calculus in the interproximal space between two incisors. Marked swelling and hypertrophy of the interdental papilla. Inflammatory resorption of the crest of the interdental bone septum. *IP*, interdental papilla; *Ca*, calculus; *R*, bone resorption. (Courtesy of Hist. Lab., Dental Inst., Univ. of Vienna.)

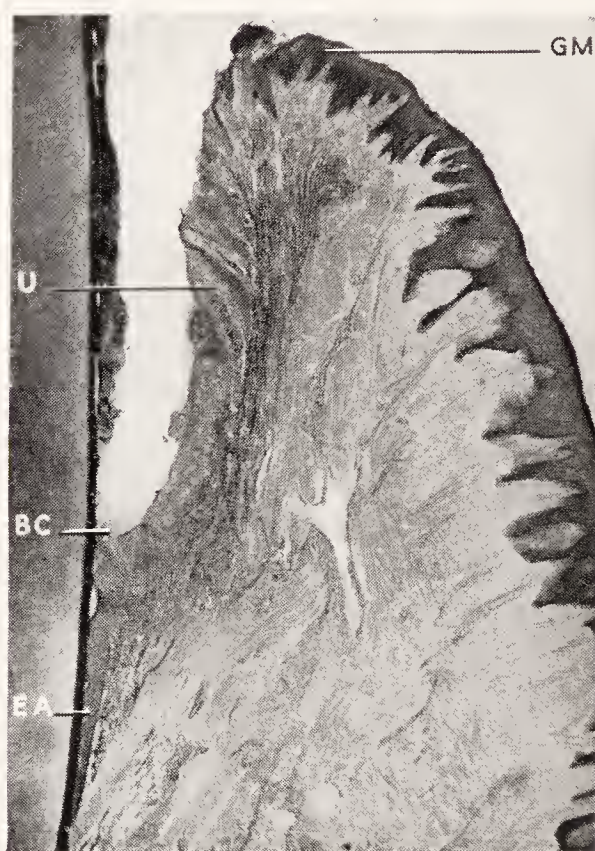


FIG. 294.—Ulceration and necrosis of the crevice epithelium. Lingual surface of an upper bicuspid. *GM*, gingival margin; *BC*, bottom of gingival crevice; *U*, ulcerated wall of the pocket. Through the breaks in the epithelium, leukocytes migrate from the subepithelial tissues into the crevice (pus formation); *EA*, epithelial attachment to the cementum.

extends as a horizontal ledge along the lingual side of the crown. The subepithelial tissue shows dense inflammatory infiltration; next to the calculus, a small ulceration has developed in the pocket epithelium through which pus is discharged into the pocket.

In the interproximal spaces, subgingival calculus is frequently accompanied by swelling and hypertrophy of the interdental papilla (papillitis, Fig. 293). If in a case like the one illustrated in Figure 293 the teeth are examined only superficially, the calculus is

easily overlooked since it is hidden by the swollen papilla and can be discovered only by means of an explorer. The subepithelial inflammation in this case has caused resorption of the crest of the interdental bone septum.

The crevice epithelium opposite subgingival calculus is usually extensively ulcerated (Fig. 294). Sometimes it is completely destroyed, and the entire surface of the pocket wall is raw and bleeds easily. After the deposits have been removed by scaling, the wall of the pocket usually becomes epithelized, and the discharge of pus stops. Only if the pocket is extremely deep does suppuration persist after scaling (paradental pyorrhea, page 316).

Gingivitis Due to Loss of Contact Point and to Caries.—Lack of proper contact, gingival caries, overhanging or defective fillings, and sharp edges of broken-down teeth are common causes of chronic marginal gingivitis, with more or less marked resorption of the alveolar crest underlying the area of inflammation. The gingival tissues may be either extensively destroyed or may be swollen and hyperplastic.

Figure 295 shows the typical result of loss of contact point caused by approximal caries on two molars. Normally, the contact point diverts the food toward the labial and lingual surface of the teeth, and thus protects the interdental papilla from injury during mastication. If the contact point is lost, food is packed against the papilla, causing destruction and inflammation of the soft tissue and resorption of the interdental septum of alveolar bone. The interdental papilla becomes concave instead of convex, the result of pressure of impacted food particles. If this condition is allowed to persist for a long time, both soft tissues and bone between the teeth may eventually be destroyed. But if the contact point is restored and further food packing prevented, the interdental tissues heal, and the papilla is regenerated. However, there is no possibility of repair in the sense that the tissues can grow back to the root surface from which they have become detached. The term healing in this connection means only that the destructive process is arrested.

Figure 296 illustrates another case of extensive inflammation and destruction of the interdental papilla between two upper molars. Both crowns have been destroyed almost to the gingival level. The interdental papilla has been transformed into a mass of necrotic tissue, exudate, and inflammatory cells; the flat surface of the interdental gingival tissues consists of degenerating, desquamating epithelium with extensive inflammation extending far into the subepithelial connective tissue. The crest of the interdental bone has

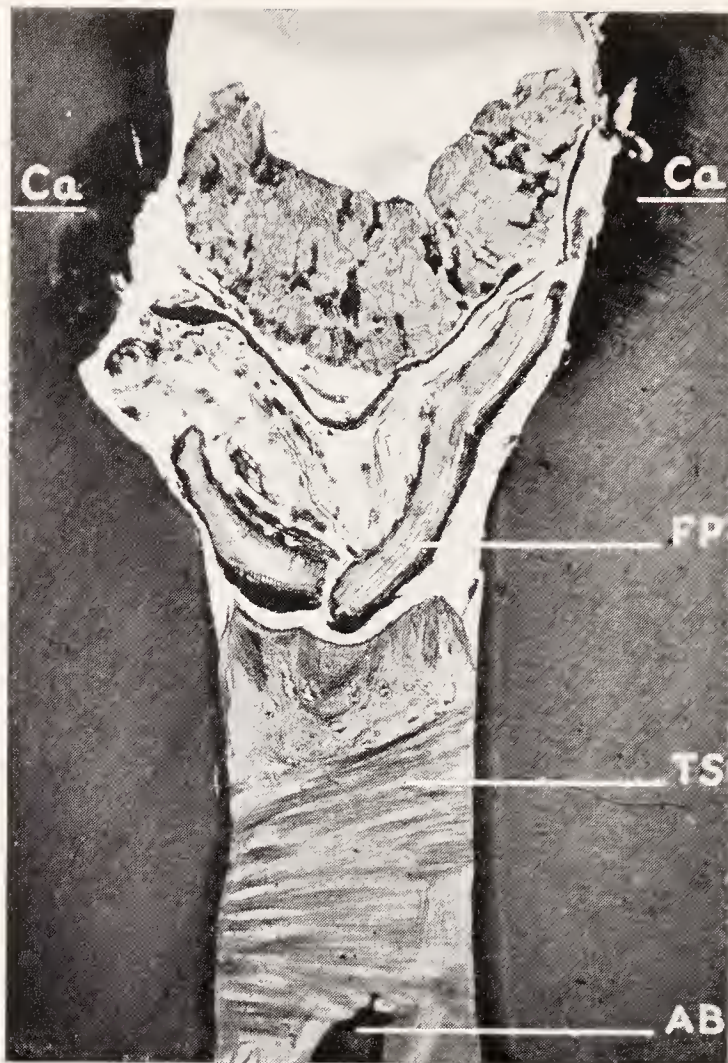


FIG. 295.—Interproximal tissues between two molars. Loss of contact point caused by approximal caries, and destruction of the interdental papilla caused by food pack. *Ca*, caries; *FP*, food particles packed between the teeth and against the papilla; *TS*, transeptal fibers; *AB*, alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

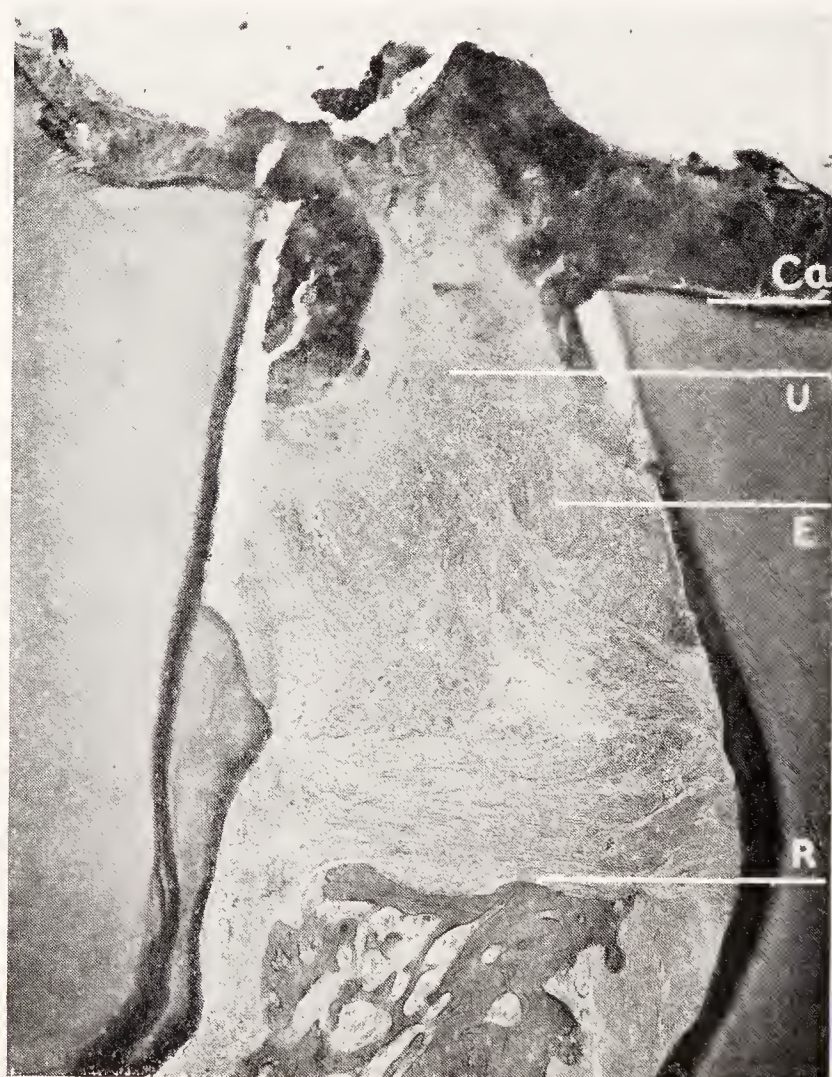


FIG. 296.—Extensive destruction of the interdental soft tissues between two broken-down upper molars. *Ca*, caries; *U*, ulcerated and necrotic surface of the interdental papilla; *E*, remnants of the gingival epithelium; *R*, resorption of the crest of the alveolar bone. (Coolidge, Jour. Am. Dent. Assn.)

been flattened and shows resorption on its surface. The deeper portions of the periodontal membrane are normal; the inflammation is confined to the marginal area.

Hyperplastic Gingivitis.—The gingival tissue may react to chronic irritation by inflammatory proliferation and hyperplasia. Sometimes this hyperplastic gingivitis is general throughout the mouth. At first there is usually a true inflammatory swelling of the gingiva. In this stage the tissue appears microscopically as granulation tissue containing varying amounts of polymorphonuclear leukocytes. If hyperplastic gingivitis persists, fibrosis of the tissues usually takes



FIG. 297.—Hyperplastic gingivitis (gingival polyp) between the roots of two broken-down upper molars. *Ca*, carious cavity; *D*, dentin of the roots; *EA*, epithelial attachment; *HG*, hyperplastic gingival tissue, showing diffuse infiltration with polyblasts. Its surface is covered with squamous epithelium.

place; the fibroblasts multiply rapidly, connective tissue is formed, the inflammatory cells disappear, and finally the tissue microscopically resembles a fibroma. Clinically this transition is characterized by decreasing hyperemia and advancing induration of the hyperplastic gingival tissue.

One form of localized hyperplastic gingivitis due to chronic mechanical irritation is the gingival polyp, a small pedunculated growth that proliferates from the interdental papilla, rarely from any other part of the gingiva. Frequently the mass of hyperplastic gingival tissue is found in a deep carious cavity (Fig. 297). Histo-

logically, the gingival polyp is granulation tissue covered with stratified squamous epithelium; the latter is attached to the neighboring teeth by an epithelial attachment. The amount of vascularization and infiltration in the hyperplastic gingival tissue depends upon the intensity of the inflammation: sometimes the epithelial covering is ulcerated, and the subepithelial tissue hyperemic and densely infiltrated; again the surface epithelium may be intact or even hornified, and the subepithelial tissue fibrous and poorly vascularized. Figure 298 shows hyperplastic gingivitis between the two roots of a deeply decayed lower first permanent molar of a child. The gingival tissue forms a mushroom-shaped growth, extending over the carious root surfaces. It is attached to the crest of the interradicular bone septum.

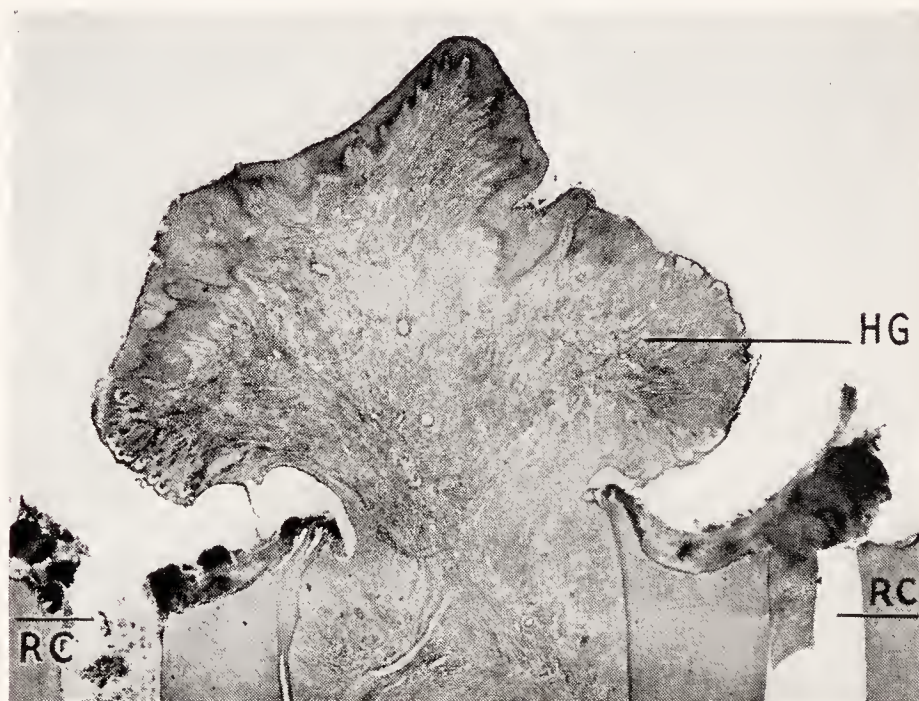


FIG. 298.—Hyperplastic gingivitis (gingival polyp) between the mesial and distal roots of a broken-down lower first molar of a child. *HG*, hyperplastic epithelized soft tissue (granulation tissue) proliferating from the interradicular septum; *RC*, root canals of the mesial and distal root of the molar.

Hornification of the Gingivæ.—The surface of the gingival epithelium is covered with a horny layer. Orban showed that whereas the mucosa of the cheeks and the vestibule does not normally become hornified, the mucous membrane surrounding the teeth and forming the gingivæ and the interdental papillæ normally has a keratinized surface. Hornification of the gingival mucosa is an important factor in the maintenance of healthy normal gingivæ; in its absence the gingivæ are abnormal, red, and swollen, and bleed easily at the slightest mechanical injury.

Figure 299 illustrates the surface of the interdental papilla of an individual, aged twenty-four years. Six months previous to the

H

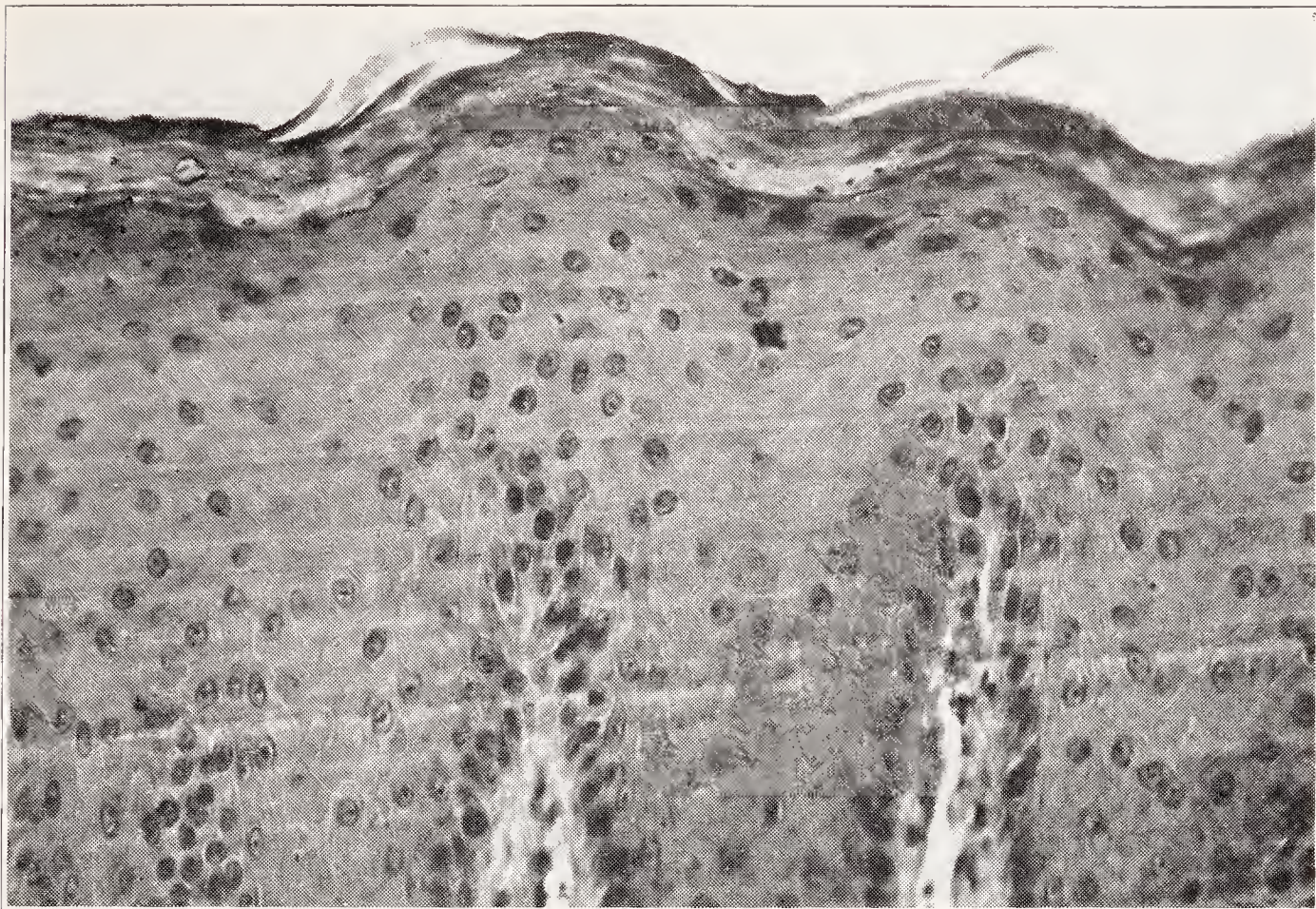


FIG. 299.—Epithelium covering an interdental papilla of an individual, aged twenty-four years. Clinically healthy, pale pink gingivæ of firm texture. Good oral care. The wavy surface of the mucosa is covered with thick horny layer, *H*. (Orban, Jour. Am. Dent. Assn.)

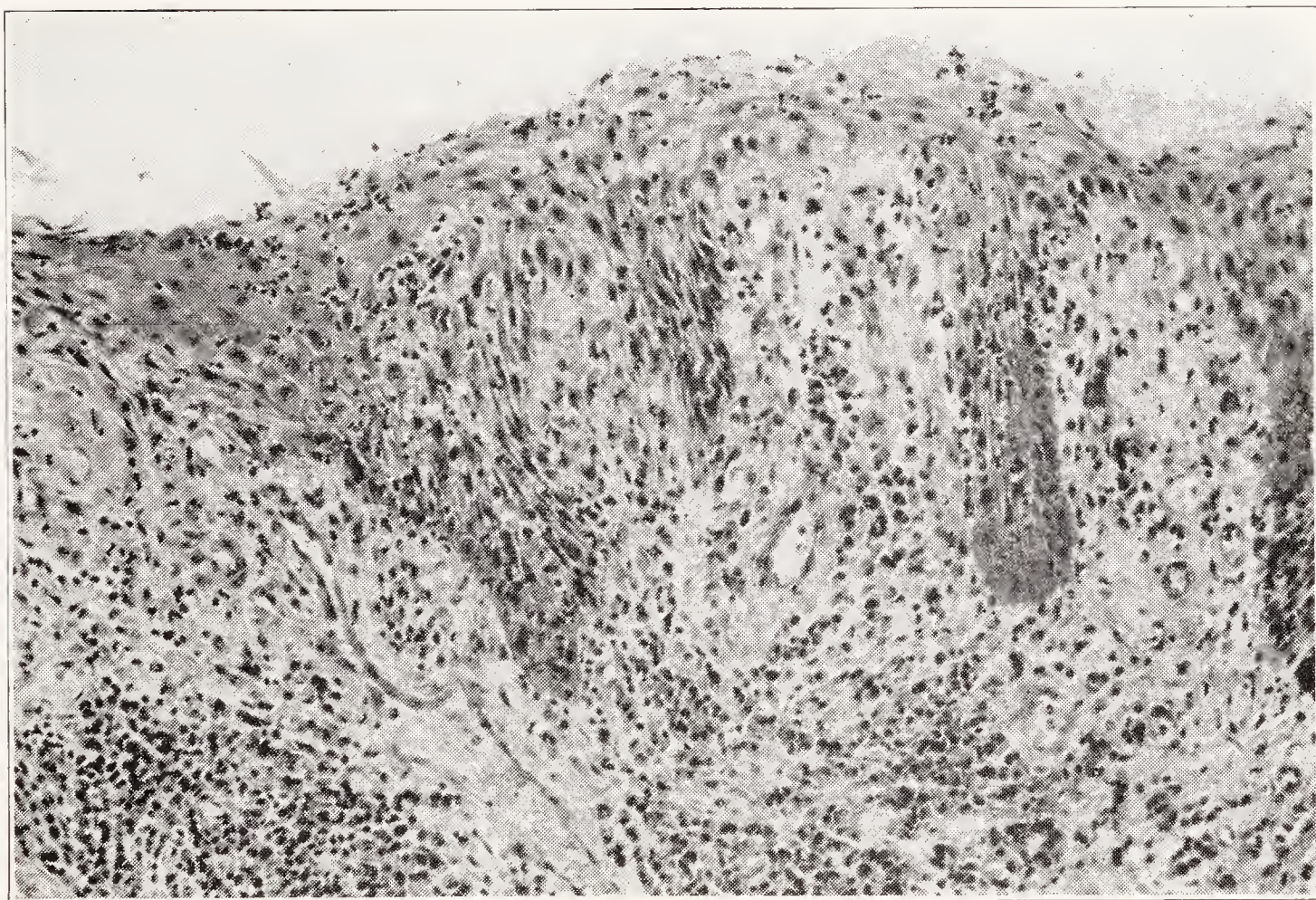


FIG. 300.—Surface of an interdental papilla of an individual, aged fifty-six years. Clinically inflamed, reddish-blue, swollen and bleeding gingivæ. Large masses of inflammatory cells are in the subepithelial connective tissue, and leukocytes migrate through the epithelium to the surface. Absence of a horny layer. (Orban, Jour. Am. Dent. Assn.)

removal of the specimen, this patient had livid, swollen, bleeding gingivæ. The teeth were scaled, and the patient was instructed in the proper use of the toothbrush. At the time of removal of the piece of gingival tissue the gingiva looked normal and healthy; it was pale pink and surrounded the teeth firmly. Histologically it shows a normal arrangement of the squamous epithelial cells with no evidence of inflammation; the surface is covered with a thick horny layer. The superficial layers of horn have been cast off.

An entirely different microscopic picture is found when clinically unhealthy looking, inflamed gingival tissues are examined. A typical specimen of this kind is reproduced in Figure 300, taken from the gingival tissue of an individual, aged fifty-six years, with swollen, reddish-blue gingivæ that bled easily. It shows degenerative changes in the epithelium, absence of a hornified layer, and extensive subepithelial infiltration with leukocytes, polyblasts, and plasma cells. The epithelium has been invaded by leukocytes, which migrate through it and are discharged into the oral cavity.

PARADENTAL PYORRHEA.

Paradental pyorrhea has been defined as a condition in which suppuration from deep pockets persists in spite of routine procedures, such as scaling and tooth brushing (Gottlieb). This definition requires some additional explanation. The evidence of suppuration alone does not justify the clinical diagnosis of paradental pyorrhea, since suppuration may occur without the presence of deep pockets; however, in the latter case the discharge is promptly arrested by the removal of the irritation. The presence of pockets alone does not substantiate the diagnosis either, since the pockets may be lined with intact epithelium, and there may be no suppuration. Only if the pockets have ulcerated walls, if the breaks in the epithelium fail to heal, and if the pockets form a habitually unclean area is the diagnosis paradental pyorrhea justified.

The etiology of paradental pyorrhea is twofold: local and systemic. Local causes for the development of paradental pyorrhea are conditions that bring about an accelerated detachment of the gingival tissues from the tooth surface without simultaneous atrophy of the detached soft tissues, thus resulting in deepening of the pocket. Such causes are mechanical injuries to the bottom of the pocket by toothpicks, loose tooth-brush bristles, or food packs, occlusal trauma with lateral stress causing injury to the periodontal tissues on the side of pressure, and unequal rate of detachment of the soft

tissues in the interproximal area, resulting in different levels of attachment.

A systemic cause for the development of paradental pyorrhea is diffuse atrophy of the alveolar bone. In this disease, deep pockets may develop rather rapidly, sometimes accompanied by loosening or pathological wandering of the teeth. The destruction of bone occurs first; it is followed by accelerated proliferation of the epithelial attachment and detachment from the root surface, producing deep pockets. The latter become secondarily infected and supuration starts. In pathological wandering, the paradental pocket develops on the side from which the drifting tooth moved; in the labial drifting of an upper incisor, the pocket develops on the lingual side of the root, in diastema formation, between the two teeth.

Figures 301 and 302 illustrate a case of paradental pyorrhea probably caused by excessive lateral stress. The specimen is an upper cuspid of a man, aged thirty-eight years. The mouth had been badly mutilated by extractions. There was a deep overbite, and the lower cuspid was almost completely covered by the upper cuspid, so that the latter was subjected to considerable lateral stress in a labial direction, as indicated by the arrow in Figure 301. The bottom of the gingival crevice had passed the cemento-enamel junction in its entire circumference. On the lingual and partly on the mesial and distal sides, the bottom of the crevice is located just rootward from the cemento-enamel junction; the crevice in these areas is shallow and healthy (Fig. 304). On the labial side, however, the tissues have become detached from the cervical third of the root surface; the gingival margin is at the same level as on the lingual side, forming a pocket 7 mm. deep. The pocket is narrow at the opening, becomes wider toward the bottom, and has the greatest width near the bottom. The epithelial tissue shows extensive inflammatory infiltration, especially near the bottom of the pocket (Fig. 303). About one-third of the labial plate of the alveolar bone has been lost by resorption. Along the mesial and distal sides of the root the bottom of the pocket extends crownward from the more apical attachment on the labial side to the cervical attachment on the lingual side.

The specimen illustrated in Figures 301 and 302 brings up the question of the treatment of such a paradental pocket. The cuspid was firm at the time of death, since the intact periodontal membrane on the entire lingual and on the greater part of the mesial, distal, and labial root surfaces was sufficient to hold the tooth firmly in place. But if such a condition is allowed to continue, more and

more of the root surface gradually loses its attachment and becomes denuded. In such a deep pocket a vicious circle develops: inflammation and suppuration destroy the tissue attachment and cause deepening of the pocket, which in turn is followed by an increase in inflammation and discharge of pus.

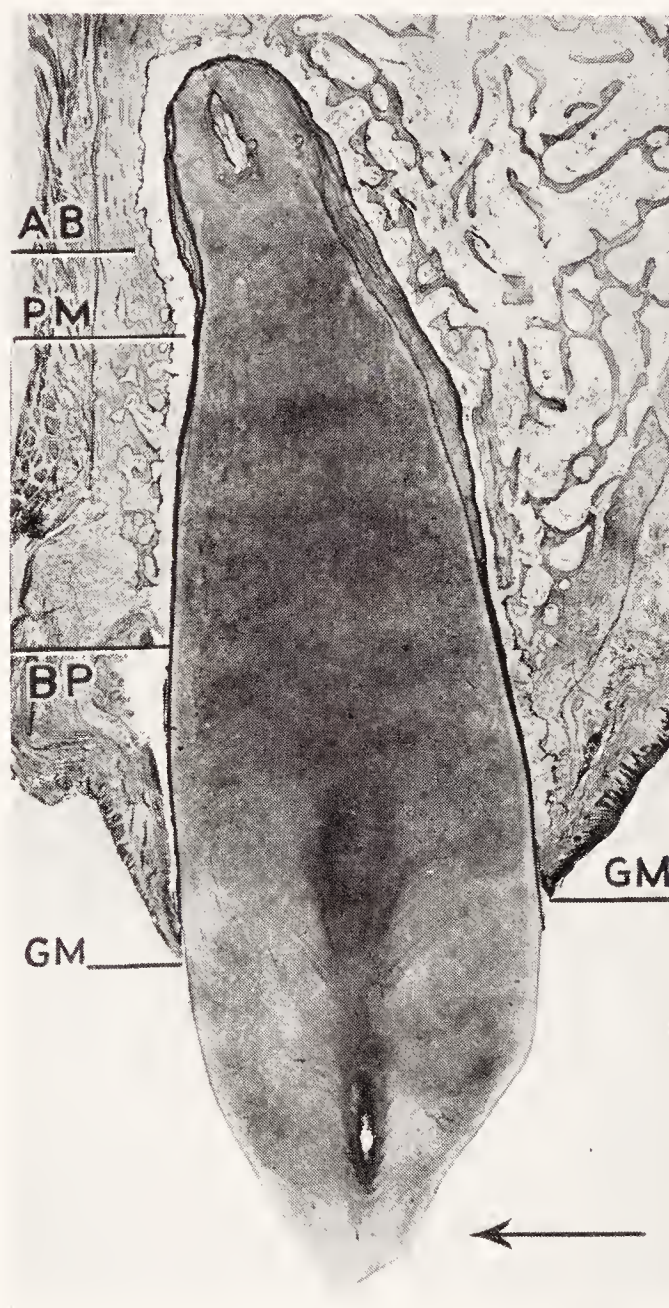


FIG. 301

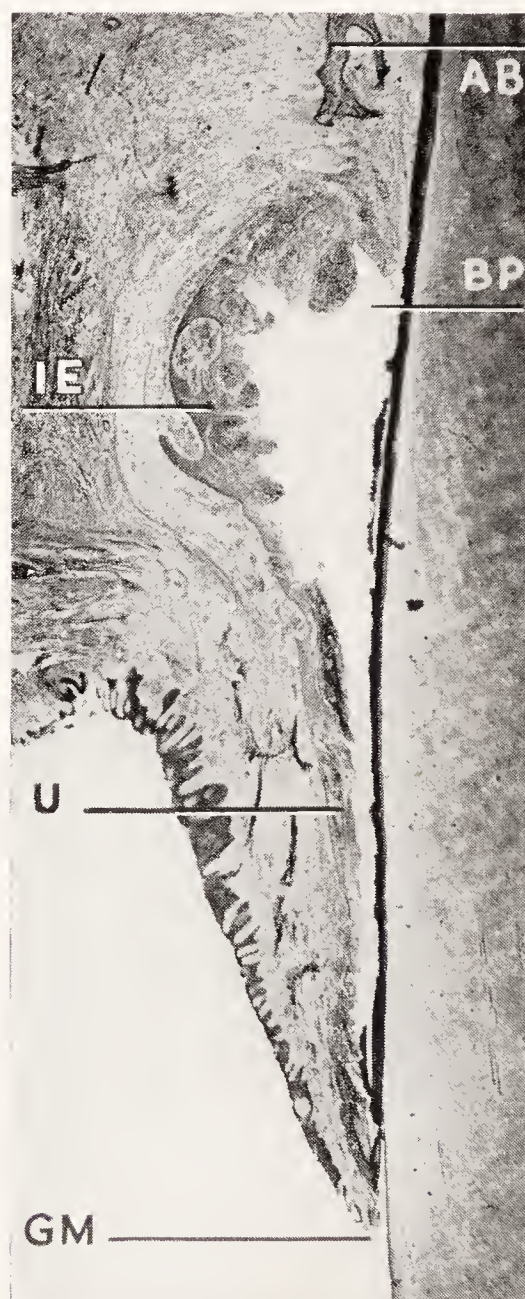


FIG. 302

FIGS. 301 and 302.—Paradental pyorrhea on the labial side of an upper cuspid. The arrow indicates the direction of the occlusal stress. On the labial surface there is a pocket of about 7 mm. in depth; on the lingual surface the depth of the crevice is zero. (See Figure 304.)

FIG. 301.—General view of the cuspid. Labio-lingual section. *GM*, gingival margin; *BP*, bottom of paradental pocket on the labial side; *PM*, periodontal membrane; *AB*, alveolar bone.

FIG. 302.—Higher magnification of the labial surface of the tooth shown in Figure 301. *GM*, gingival margin; *U*, ulcerated wall of the pocket; *BP*, bottom of the pocket; *IE*, inflammatory proliferation of epithelium; *AB*, alveolar bone.

The therapy for a condition like the one illustrated in Figures 301 and 302 consists of the removal of the outer wall to the bottom of the pocket. All the tissue between *GM* and *BP* should be removed

in order to establish clean conditions and prevent progressive destruction. This can be done by excision, cauterization, packing, or by a combination of any of these methods. If properly carried out, such therapy will result in a crevice of zero depth on the level of *BP*. The root surface between *BP* and *GM* will be exposed. Any etiological factors, such as occlusal overstress, have to be corrected to prevent further pocket formation.

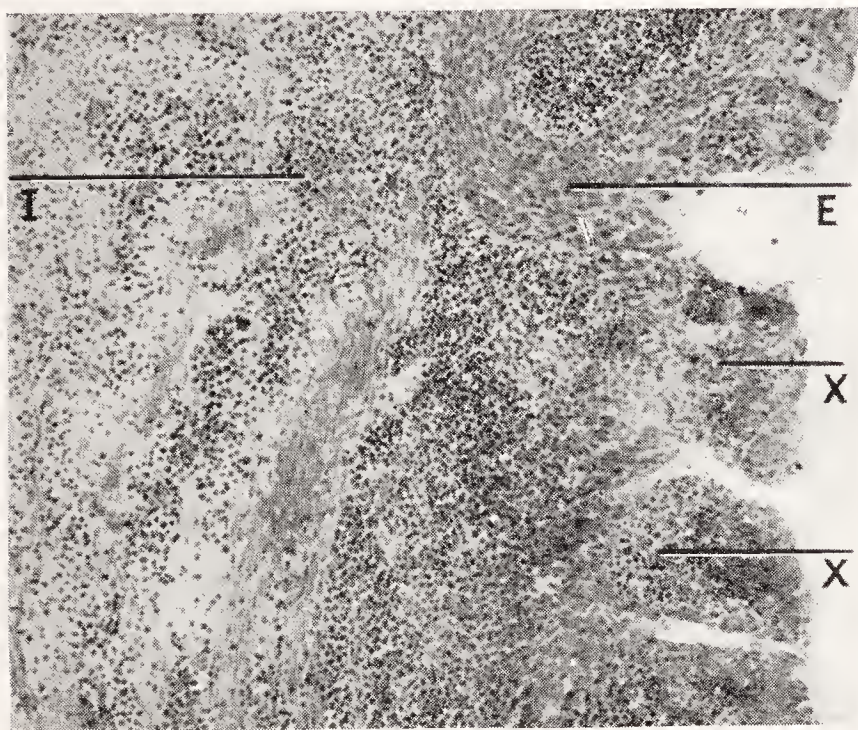


FIG. 303

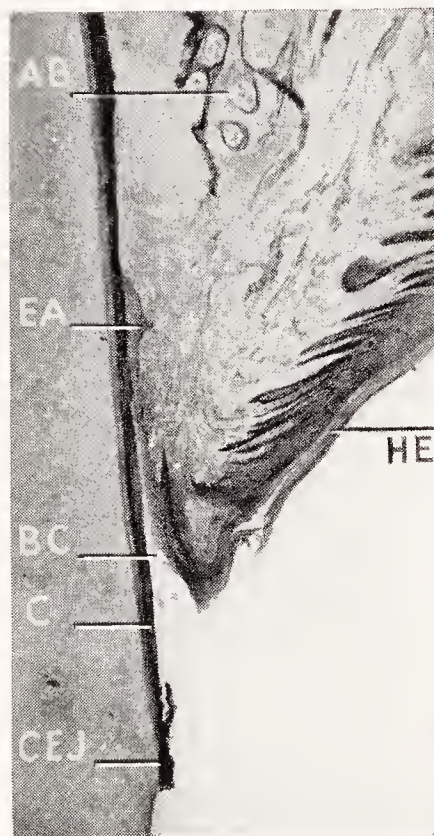


FIG. 304

FIG. 303.—High magnification of the wall of the paradental pocket in Figure 302. *I*, subepithelial accumulation of inflammatory exudate cells; *E*, remnants of the crevice epithelium; *X*, breaks in the epithelium, through which leukocytes migrate into the pocket.

FIG. 304.—Higher magnification of the gingival margin on the lingual side of the cuspid in Figure 301. Normal gingival crevice. The depth of the crevice is practically zero. *CEJ*, cemento-enamel junction; *C*, cementum; *BC*, bottom of gingival crevice located on the cementum; *EA*, epithelial attachment to the cementum; *HE*, hornification of the gingival epithelium; *AB*, alveolar bone.

Bone Changes Under Pyorrhea Pockets.—The condition of the alveolar bone underlying periodontal pockets is illustrated in Figures 305 and 306. The specimen is an upper central incisor of a man, aged fifty-five years. On the lingual side of the crown a pocket extends over more than one-half of the root. The epithelial lining of the pocket is ulcerated, and the subepithelial connective tissue densely infiltrated with inflammatory exudate cells (Fig. 306). The bone facing the pocket is covered with osteoclasts (R_1); many Howship's lacunæ indicate the activity of the resorptive process. Similar changes are also found in the adjacent marrow spaces (R_2).

These bone changes are characteristic of the condition found by the author in numerous specimens of pyorrhea pockets. Never is exposed or necrotic bone observed without there being a layer of connective tissue between pocket and bone; and the latter, although inflamed, is always vital. From these findings the author



FIG. 305.—Bucco-lingual section through upper central incisor. Age, fifty-five years. *P*, periodontal pocket of 7 mm. depth on lingual side of root; *R*, resorption of lingual alveolar bone crest; *SD*, secondary dentin in pulp chamber. (Kronfeld, Jour. Periodontology.)

is convinced that the bone underlying pyorrhea pockets is in a state of low-grade, chronic osteitis with osteoclastic bone resorption, but is not necrotic.

Paradental Pyorrhea on the Approximal Surfaces of Teeth.—One of the causes for the development of paradental pockets is a

different amount of detachment of the soft tissues on the surfaces of two neighboring teeth. If the detachment on one root is rather far advanced and on the tooth next to it the tissues are still attached

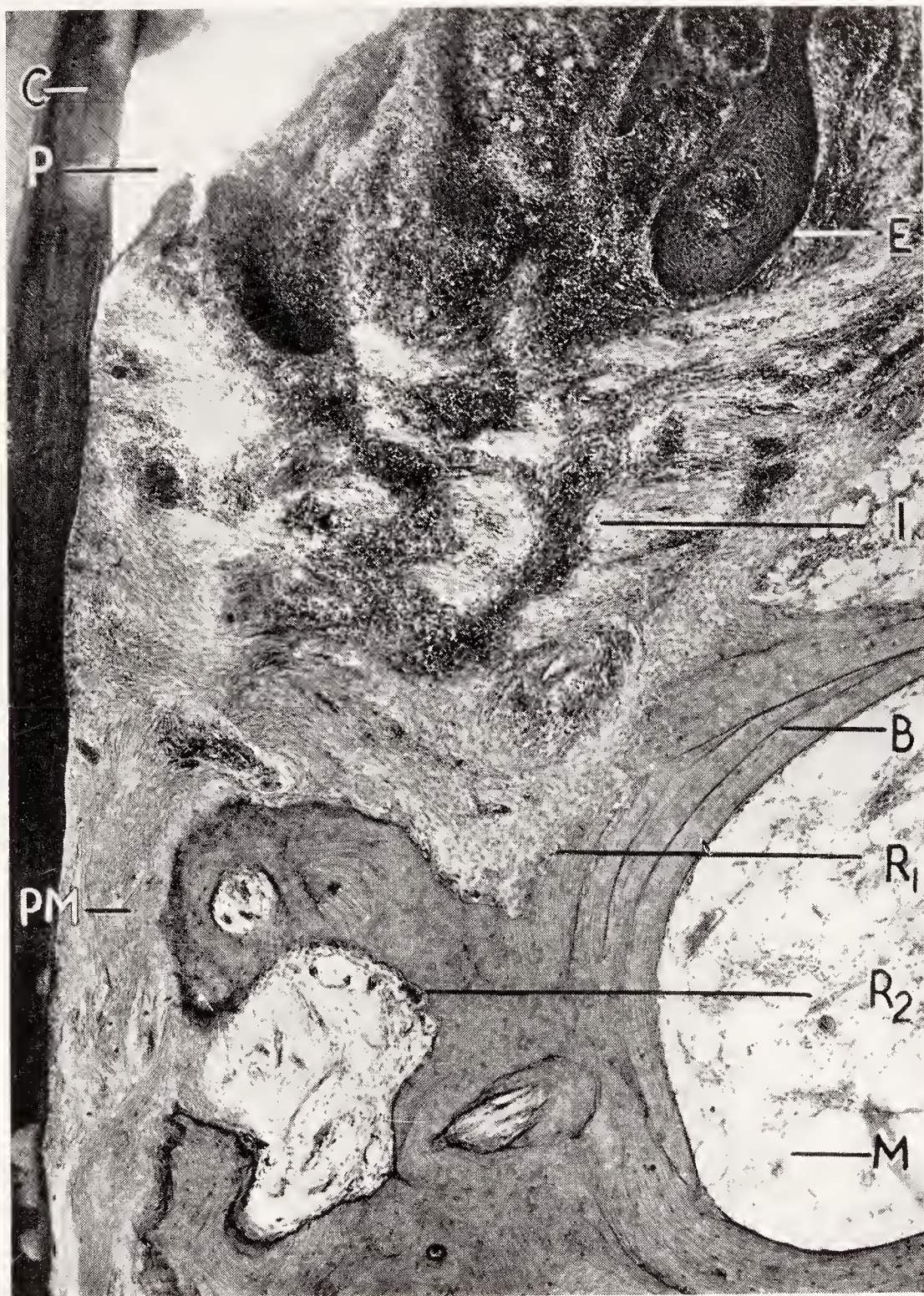


FIG. 306.—Higher magnification of bottom of pocket in Figure 305. *C*, cementum; *P*, bottom of pocket; *E*, inflammatory epithelial proliferation; *I*, infiltrated connective tissue between pocket and bone; *R*₁, resorption of bone adjacent to the inflamed connective tissue; *R*₂, resorption of bone on the wall of a marrow space; *M*, marrow space; *PM*, periodontal membrane; *B*, bone lamellæ. (Kronfeld, Jour. Periodontology.)

near the cemento-enamel junction, shrinkage and atrophy of the interdental papilla cannot keep pace with the detachment at the bottom of the crevice. The result is the development of a paradental pocket. Figure 307 illustrates such a condition in the interdental

space between two lower incisors. On the right side the pocket is shallow; the papilla is normal and covered with a keratinized layer. On the left side the bottom of the pocket is located at a considerably lower level; on this side the wall of the papilla is ulcerated, and pus is being discharged into the pocket.

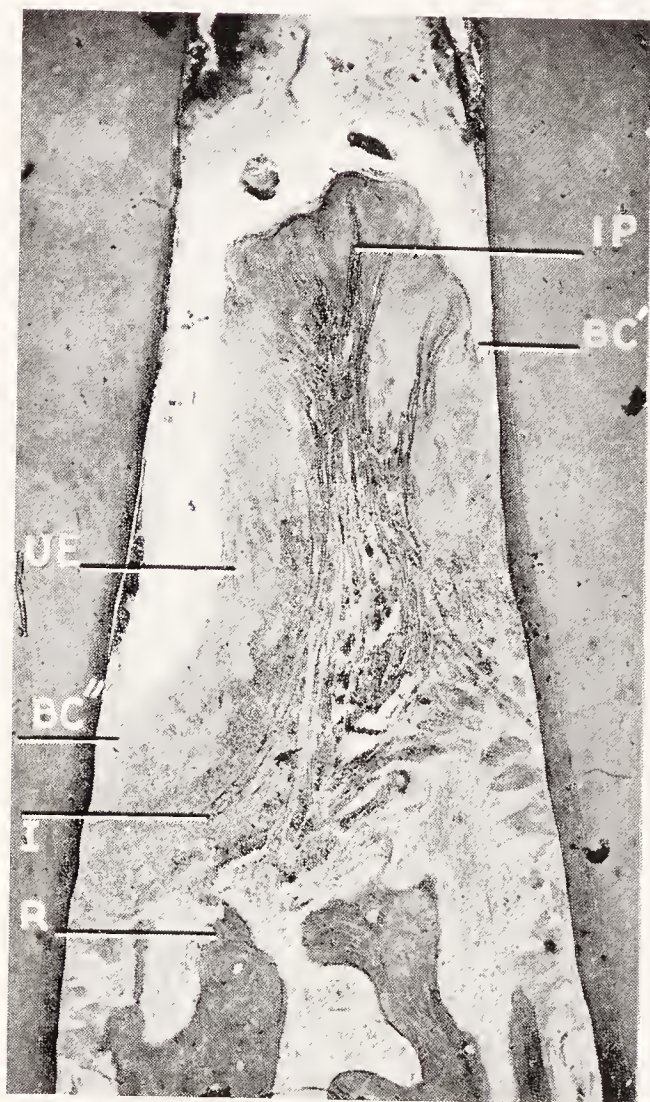


FIG. 307.—Paradental pyorrhea in the interproximal space between two lower incisors. Deep pocket caused by a different level of attachment of the interdental papilla on the two teeth. *IP*, interdental papilla; *BC'*, bottom of the crevice on the right tooth; shallow crevice, intact crevice epithelium. *BC''*, bottom of the crevice on the left tooth; deep crevice with ulcerated crevice epithelium, *UE*; *I*, subepithelial inflammation; *R*, resorption of the alveolar bone at the crest of the interdental septum.

In deep paradental pockets with narrow openings, acute purulent inflammation is occasionally observed. This condition is caused by accumulation and retention of infective material in the pocket and is called paradental abscess or lateral abscess; it must not be confused with periapical abscesses from infected pulpless teeth.

The Problem of Reattachment.—The clinical observation of deep pockets has quite naturally encouraged attempts to bring about a reattachment of the detached gingival tissues to the root surface. Clinicians and pathologists have reported reattachment of the gingival tissue in deep pockets following treatment and medication. However, the author is of the opinion that such reports have to be considered with a scepticism based upon practical as well as theoretical considerations.

The difficulty in diagnosing reattachment after treatment of a deep pocket lies in the fact that the wall of the pocket has a

tendency to shrink; as a result, a pocket may appear shallower after treatment than before, but the reduced depth may be due to contraction of the pocket wall and not to reattachment. The same process of shrinkage can also bring about a close adaptation of the formerly loose, flabby pocket to the root surface, which resembles reattachment and makes exploration difficult. In order to prove

that the tissue has actually become reattached to the root surface, it would be necessary to measure the exact distance to the bottom of the pocket, before and after the treatment, from a fixed point on the tooth surface, as, for instance, from a filling; reattachment may be claimed only if such measurements show that the distance from the bottom of the pocket to the margin of the filling has actually become shorter. To the author's knowledge such measurements have not yet been reported.

The second reason for the author's scepticism about the possibility of reattachment is that the root surface, once it has become denuded and exposed over a long period of time, is in a biological sense a dead surface. Under these circumstances, deposition of new layers of cementum, without which a functional reattachment is impossible, is unlikely and up to this time has not been demonstrated.

Another complication is the presence of the epithelial attachment to the root surface. As long as the wall of the pocket is covered with epithelium, reattachment of connective tissue is impossible. Even if it were possible to remove all epithelium to the bottom of the pocket, beyond this point there is still the epithelial attachment from which new epithelium can proliferate and interfere with cementum formation and connective tissue reattachment.

All these factors contribute to make the conditions for reattachment relatively unfavorable. Therefore, the author believes that even though there may theoretically be a possibility of reattachment, the practical value of the therapeutic methods based upon this possibility is rather questionable. But he also wishes to emphasize his willingness to revise his opinion on this subject if clinical proof of reattachment can be established by accurate measurements and if deposition of cementum and attachment of functioning periodontal fibers can be demonstrated on a portion of the root surface that previously was exposed in the pocket. In the few histological specimens of human teeth in which reattachment has been described, either there was merely an attachment of epithelium to the root surface, which is of no value from a functional point of view, or it was not at all certain whether the supposedly reattached tooth surface had ever been exposed.

Radiographic evidence of bone regeneration following treatment of pyorrhea pockets has frequently been observed, but has no direct relation to reattachment. The routine radiograph does not show the amount of attachment or detachment of the soft tissue, but merely the amount of bone. Bone destruction and soft tissue detachment are not necessarily correlated; there may be extensive bone

destruction without accompanying soft tissue detachment. In such cases bone regeneration is possible, but this does not prove a reattachment of formerly detached soft tissues.

ALVEOLAR ATROPHY CAUSED BY EXCESSIVE OCCLUSAL STRESS.

Loss of alveolar bone because of excessive occlusal stress is most likely to occur in mouths in which many teeth are missing and in which no restorations have been made to replace the lost teeth. In such mouths the remaining teeth have to endure the entire masticatory force that was formerly distributed over all thirty-two teeth. Another condition that is likely to be accompanied by excessive occlusal stress is the tipping of a tooth after the extraction of its neighbor, for example, the forward tipping of a lower second molar following extraction of the first molar. Such a tipped tooth is subjected to considerable lateral stress, which leads much more easily to injury of the periodontal tissues than vertical stress.

Under the above-mentioned circumstances teeth may become loosened because of pressure atrophy of the alveolar bone. Whether or not this actually happens depends upon the individual tissue resistance. Some patients, with only a few remaining teeth and these often in unfavorable occlusal conditions, use these overloaded teeth for decades without damage. The microscopic examination of teeth of this kind reveals a reënforcement of the periodontal membrane and supporting bone, indicating great resistance and successful adaptation to the increased functional requirements. In other patients one tooth after the other becomes loose and finally is lost because of excessive occlusal stress. Such teeth microscopically show various forms of tissue injury, necrosis of the periodontal membrane, root resorption, and extensive resorption of the alveolar bone without reparative or regenerative bone changes.

An example of loosening of a tooth under excessive occlusal stress is illustrated in Figure 308. The specimen is a lower second bicuspid. The incisors, first bicuspid, and all molars were missing in this mandible. In the upper jaw all teeth were present. The closing of the bite and the loss of the first bicuspid forced the crown of the second bicuspid toward the mesial side until, at the time of death, the tooth had been tipped about 30 degrees from its normal vertical position. The radiograph shows that the alveolar bone has been lost except for a small area at the apex. The histological sections reveal even more extensive destruction of bone. The radiographic appear-

ance of bone at the apex apparently is the result of a projection of the bone on the buccal and lingual side of the mandible over the apex; in a central section through the tooth, there is no alveolar bone whatsoever around the apex. The bicuspid is embedded in fibrous connective tissue that is attached to the apical two-thirds of the root surface. This specimen illustrates that no definite relationship exists between location of the bottom of the pocket and position of the alveolar crest. The bone was resorbed as a result of excessive occlusal stress, but the detachment of the periodontal soft tissue did not follow the recession of the bone.



FIG. 308.—Mesial tipping of a lower right second bicuspid after the loss of the neighboring teeth. Extensive destruction of the alveolar bone. The radiograph of the specimen in the upper left corner shows the amount of bone destruction. A mesio-distal section through the jaw shows that the bicuspid is embedded only in fibrous connective tissue. Notice the location of the bottom of the gingival crevice on the root surface of the bicuspid. Despite the extensive loss of alveolar bone, about two-thirds of the surface of the anatomical root are still in organic connection with the surrounding tissues. The periodontal tissues of the lower cuspid are intact.

This observation shows that the usual procedure of diagnosing “pockets” from a radiograph is not correct. The location of a pocket on the tooth surface is determined by the soft tissue attachment, not by the bone; a radiograph can reveal a pocket only after a radiopaque material (for instance, a guttapercha point) has been introduced into the pocket and is radiographed with the tooth. The only thing that can be seen in the routine radiograph is the amount of bone;

the supposition that loss of bone and formation of a pocket necessarily go hand in hand is erroneous.

In making a prognosis for a tooth that has been loosened by excessive occlusal stress, the attachment of the soft tissues must be considered first and the condition of the bone second. If a loosened tooth is still attached to the periodontal tissues by a considerable area of its root surface, there is a possibility of bone regeneration if the harmful overstress is eliminated by artificial restorations. If, however, the soft tissues have become extensively detached from the root and a deep pocket has developed, the prognosis for the tooth is unfavorable, since alveolar bone can regenerate only where connective tissue is still united with the root surface.

DIFFUSE ATROPHY OF THE ALVEOLAR BONE.

The term "diffuse atrophy of the alveolar bone" to describe loss of alveolar bone with clinical symptoms of loosening and drifting was introduced in 1923 by Gottlieb. Since that time, several investigators studying the same clinical condition have used new terms, such as complex periodontitis (Box) and genuine paradentosis (Becks). The clinical aspect of diffuse atrophy has been described (see page 315); the microscopic findings will be discussed here.

Typical diffuse atrophy of the alveolar bone is by no means a common disease. Histological material for its study is therefore difficult to obtain. The best known case is the one studied by Gottlieb (Fig. 309). The patient, a man, aged twenty-two years, had died of pneumonia following influenza and general exhaustion. All teeth microscopically show resorption of both root surface and alveolar bone, widening of the periodontal space, and transformation of the fibrous periodontal membrane into loose, vascular connective tissue without functional orientation (Figs. 310 and 311). The resorptive processes and the changes in the structure of the periodontal membrane are confined to the apical two-thirds of the roots; the gingival third of the roots and the interdental soft tissues are intact. The individual being a young adult, the gingival crevice is still on the enamel; there is very little subepithelial inflammation in the gingival tissue.

Resorption of the alveolar bone and widening of the periodontal membrane are clinically manifested by loosening of the teeth. The destruction of the periodontal fibers and their transformation into loose connective tissue explain the displacement of the teeth known as pathological wandering. If the destructive process takes place



FIG. 309



FIG. 310

FIG. 309.—Diffuse atrophy of the alveolar bone. Man, aged twenty-two years. Evidence of resorption on the roots, especially in the apical portion. Loss of the normal fibrous structure of the periodontal membrane and resorption of the adjacent alveolar bone. The general view of the upper central and part of the lateral incisor shows the location of the resorptions, *R*, at the apices of both teeth. The remaining portion of the periodontal membrane appears normal; the gingival tissues are intact; no deposits are present. The bottom of the crevices is located on the enamel. (Gottlieb, Jour. Am. Dent. Assn.)

FIG. 310.—Higher magnification of the apex of the central incisor. *RR*, resorption of the root surface; *CT*, loose vascular connective tissue replacing the fiber bundles of the periodontal membrane; *BR*, bone resorption; *P*, pulp strand passing through apical foramen. Notice the irregular widening of the periodontal space around the entire apical third of the root.

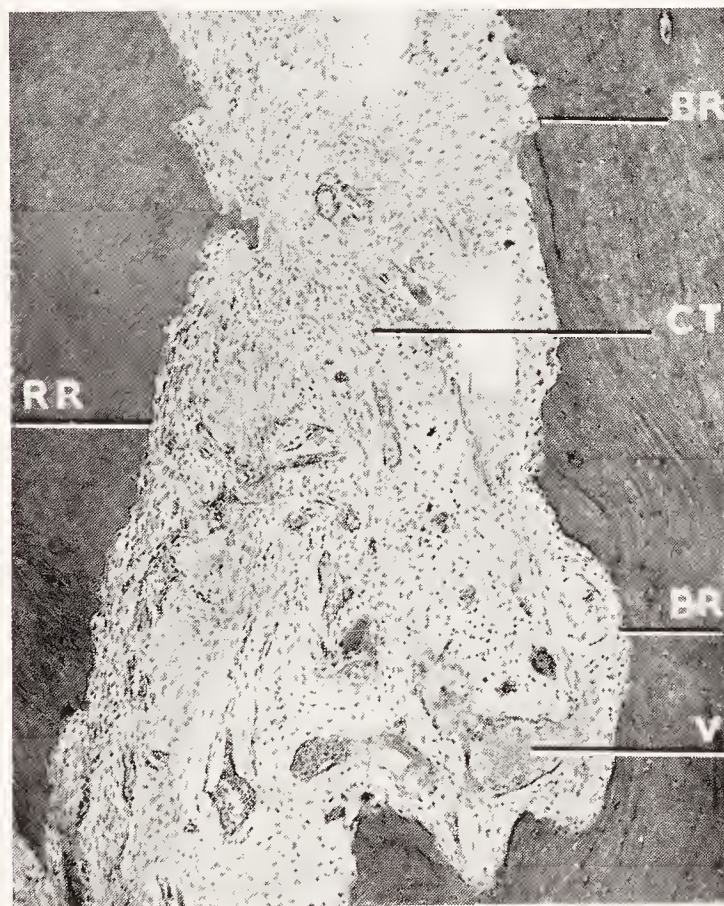


FIG. 311.—High magnification of a portion of the root surface near the apex in Figure 310. *RR*, resorbed dentin surface; *CT*, loose connective tissue; *V*, blood-vessels; *BR*, bone resorption.

in the apical portion of a tooth, the loss of the fibers and the tension of the newly formed connective tissue in this area cause elongation (extrusion, pathological wandering in vertical direction). If the changes take place on one side of the root, the tooth is displaced toward the opposite side (pathological wandering in horizontal direction, drifting); later a pocket usually develops on the side of the root on which bone was resorbed.

Gottlieb offers the following hypothesis to explain this generalized root and bone resorption: In diffuse atrophy, the vitality of the root surface has been impaired in some way, resulting in loss of attachment of the periodontal fibers to the root surface. Subsequently the bone, receiving no more stimuli in the damaged area, is resorbed and becomes atrophic. In the case illustrated here, influenza may have been the cause for the resorption of the root surface; in other cases diabetes (Figs. 285 and 286) or some other systemic or metabolic disturbance may play a rôle in the etiology. Recently Boyle, Bessey, and Wolbach pointed out the similarity between the pathological changes in the periodontal tissues of animals with experimental scurvy and those in the human periodontal tissues in diffuse alveolar atrophy. From this similarity they suggested that a low content of ascorbic acid (vitamin C) in the blood may be a contributing factor to the diffuse atrophy type of periodontal disease.

Occlusal trauma is of secondary importance in the development of diffuse atrophy. Once a tooth has begun to change its position as the result of pathological wandering, it will sooner or later be subjected to excessive occlusal stress which, in turn, accelerates its loosening. Therefore, the occlusion must be carefully checked, and whenever a tooth shows a tendency to drift or elongate, it should be ground out of occlusion to prevent secondary occlusal overstress.

The prognosis of diffuse atrophy is doubtful. Sometimes the widening of the periodontal membrane and the loss of alveolar bone continue until the tooth is lost. Again, the pathological process may come to a temporary or permanent standstill; new cementum is formed on the root surface, new periodontal fibers are built, and the alveolar bone is regenerated. No such repair is possible if deep pockets have already formed, because the presence of a pocket prevents the deposition of new cementum on the root surface.

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CHAPTER XIV.

INFLUENCE OF FUNCTION UPON TEETH AND SURROUNDING STRUCTURES.

ARRANGEMENT AND FUNCTION OF THE FIBERS OF THE PERIODONTAL MEMBRANE.

THE root of a tooth is attached to its socket by groups of fiber bundles which constitute the periodontal membrane or dento-alveolar ligament. The ends of these fibers are embedded in the cementum and the alveolar bone, respectively; the embedded portions are called Sharpey's fibers. The fiber bundles of the periodontal membrane are divided into five groups according to their

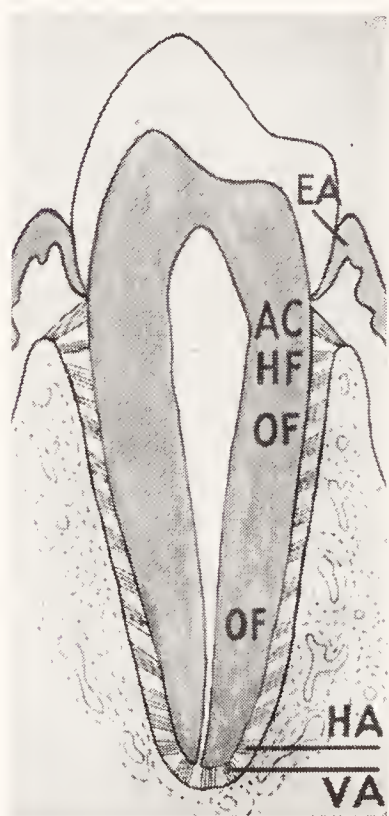


FIG. 312

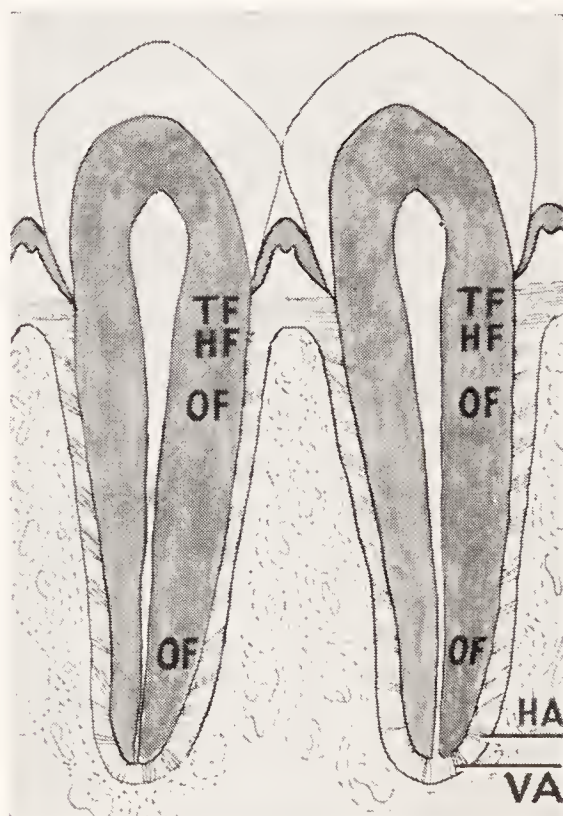


FIG. 313

FIGS. 312 and 313.—Fiber groups of the periodontal membrane.

FIG. 312.—Bucco-lingual section through lower first bicuspid. *EA*, epithelial attachment; *AC*, alveolar crest fibers; *HF*, horizontal dento-alveolar fibers; *OF*, oblique fibers; *HA*, apical fibers, horizontal group; *VA*, apical fibers, vertical group.

FIG. 313.—Mesio-distal section through lower first and second bicuspid. *TF*, transeptal fibers; *HF*, horizontal dento-alveolar fibers; *OF*, oblique fibers; *HA*, apical fibers, horizontal group; *VA*, apical fibers, vertical group.

course and distribution: transeptal fibers, alveolar crest fibers, horizontal dento-alveolar fibers, oblique dento-alveolar fibers, and apical fibers (Figs. 312 and 313). An additional, sixth group of fibers, the free gingival fibers, run from the alveolar margin to the gingiva; they do not contribute to the mechanical retention of the root in the socket.

Transeptal Fibers.—The transeptal fibers are confined to the area between two adjacent teeth; they run from the cementum of one tooth in a more or less horizontal direction across the crest of the interdental septum to the cementum of the next tooth (Fig. 314). Their function is to maintain the mesio-distal relationship between neighboring teeth.

Alveolar Crest Fibers.—The alveolar crest fibers are a rather variable group of fibers. In some teeth they are well developed, in others entirely missing. They run from the crest of the alveolus

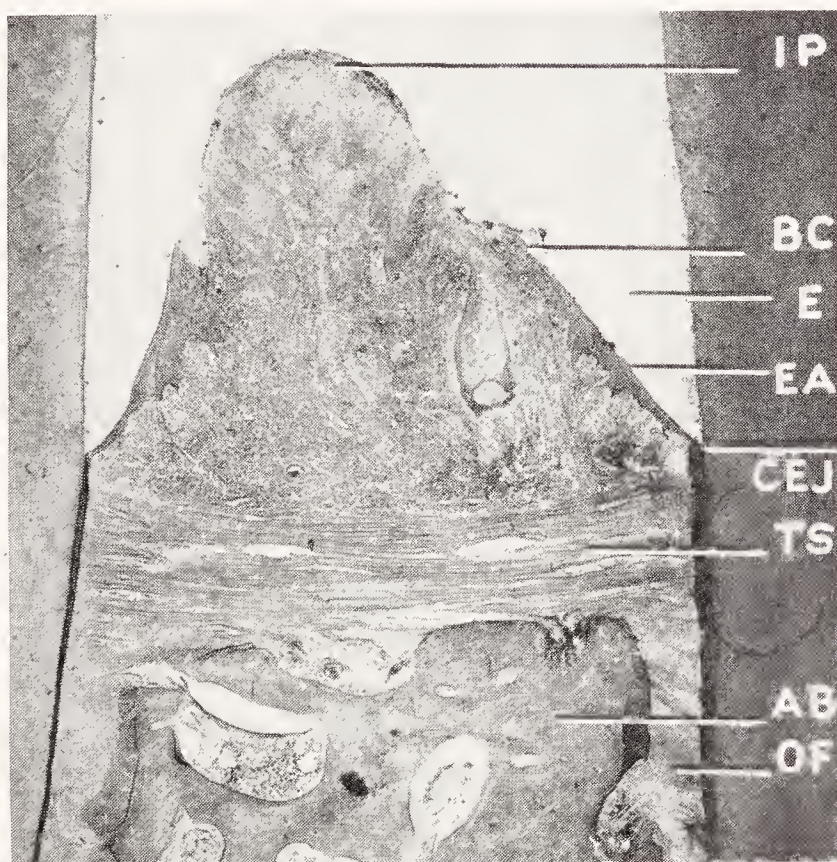


FIG. 314.—Transeptal fibers between upper central and lateral incisors. *TS*, transeptal fibers; *AB*, alveolar bone; *OF*, oblique fibers; *CEJ*, cemento-enamel junction; *E*, enamel; *EA*, epithelial attachment to the enamel; *BC*, bottom of gingival crevice; *IP*, interdental papilla.

to that area of the cementum which is bordered crownward by the deepest point of the epithelial attachment and rootward by the crest of the alveolar bone (Fig. 315). Their function is identical to that of the horizontal dento-alveolar fibers described in the next paragraph.

Horizontal Dento-alveolar Fibers.—Horizontal dento-alveolar fibers are found around the entire tooth, running horizontally from the alveolar margin to the cementum (Fig. 315). These fibers were originally called the “circular ligament of the tooth.” Their function is to prevent displacement by lateral stress.

The location of transeptal, alveolar crest, and horizontal dento-alveolar fibers on the tooth surface varies according to age. In

young individuals all of these fibers are found near the cemento-enamel junction. With advancing age, together with the proliferation of epithelium along the root surface and the atrophy of the alveolar crest, they are located farther rootward. A typical example of this change in location is illustrated in Figure 281, which shows the transeptal fibers in their relationship to teeth and bone in a case of considerable gingival recession and exposure of the root surface.

Oblique Dento-alveolar Fibers.—The oblique dento-alveolar fibers comprise the bulk of the fibers that form the periodontal membrane. Their arrangement can be understood best by comparing

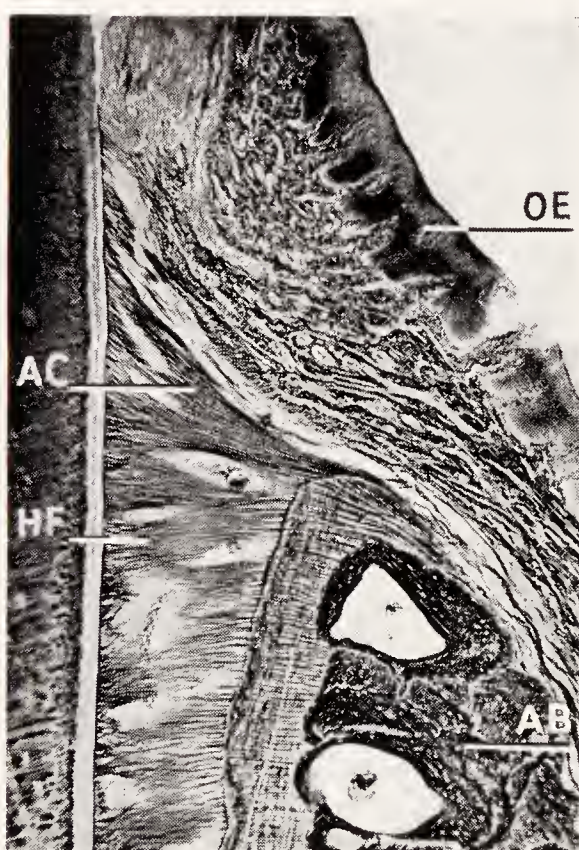


FIG. 315.—Alveolar crest fibers and horizontal dento-alveolar fibers on the lingual side of an upper second bicuspid. Silver staining. *AB*, alveolar bone; *AC*, alveolar crest fibers; *HF*, horizontal dento-alveolar fibers; *OE*, oral epithelium.

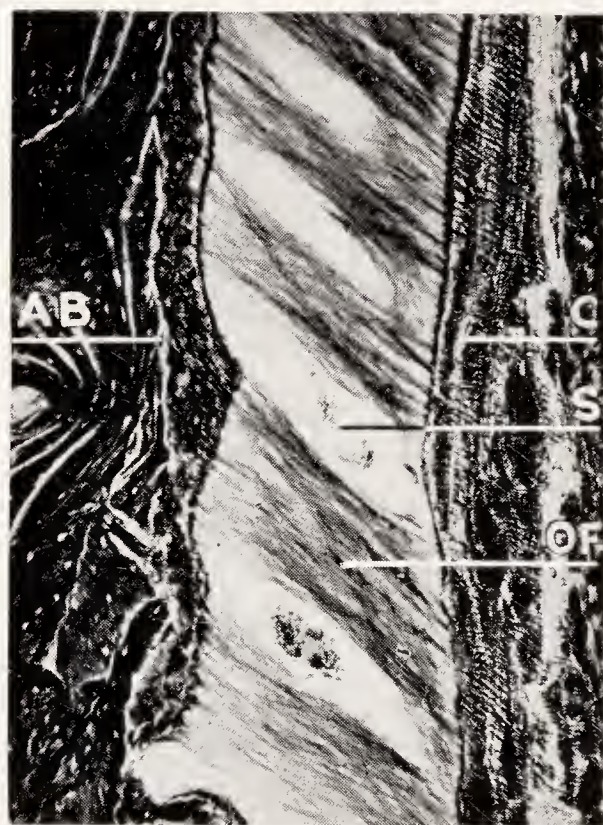


FIG. 316.—Oblique dento-alveolar fibers. Longitudinal section through the root. Silver staining. *C*, cementum; *OF*, oblique fibers; *S*, spaces between the fiber bundles containing the blood-vessels and nerves of the periodontal membrane; *AB*, alveolar bone.

longitudinal and horizontal sections. In a longitudinal section through the root, the oblique fibers run apically from the inner plate of the alveolus to the root surface at an angle of about 45 degrees (Fig. 316). The fibers form strong, dense bundles, between which are narrow spaces for the periodontal blood-vessels and nerves. In a horizontal section through the root, the oblique fibers radiate fanwise from prominent points or single trabeculae of bone on the inside of the alveolus to the surface of the cementum.

The function of the oblique fibers is to attach the root to its

socket and to counteract vertical stress. They suspend the root in the alveolus, thus transforming occlusal pressure into pull upon the alveolar bone. Bone is much better able to withstand pull than pressure: increased pull leads to functional hypertrophy of bone; increased pressure causes bone resorption. Because of their arrangement, the oblique fibers transmit the pressure of mastication to the bone as pull.

Apical Fibers.—The fibers around the apex can be subdivided into two minor groups: horizontal and vertical apical fibers. Both frequently have a more or less rudimentary development, and in

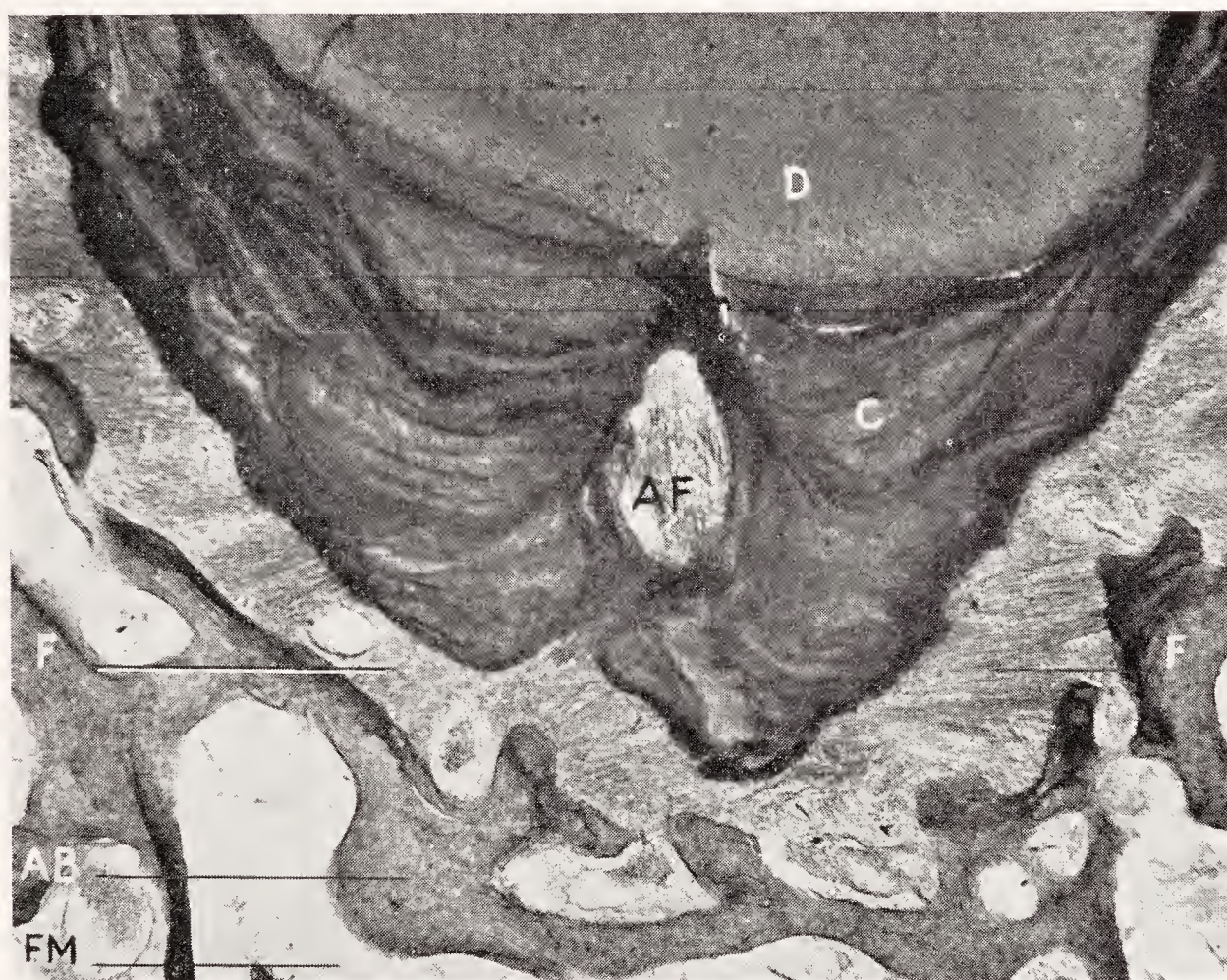


FIG. 317.—Apical fibers of the periodontal membrane. Apex of a lower cuspid. *D*, dentin; *C*, cementum; *AF*, apical foramen; *F*, apical fibers; *AB*, alveolar bone; *FM*, fat marrow.

some specimens are entirely missing. The horizontal apical fibers run in a horizontal direction from the apex of the tooth to the bone; they form a counterpart to the horizontal dento-alveolar fibers, and their function is the same as that of the latter. The vertical apical fiber bundles run vertically from the root end to the bottom of the alveolus, thus preventing lateral displacement of the apical region of the tooth. These fibers are found only in adult teeth with fully developed root ends (Fig. 317).

All fiber bundles of the periodontal membrane consist of col-

lagenous fibers; there are no elastic fibers present. In a state of complete relaxation, the bundles assume a slightly wavy course; minute movements of the tooth are possible by stretching the S-shaped fibers into straight ones.

MASTICATORY FORCES EXERTED UPON HUMAN TEETH.

The masticatory forces may be divided into two main types: vertical (axial) stress and horizontal (lateral) stress.

In mastication the teeth are exposed to a force that is the resultant of vertical and horizontal stress; whether the vertical or the horizontal component prevails depends upon the condition in the individual mouth and upon the different masticatory movements. Some teeth are subjected to an almost purely vertical or purely horizontal stress.

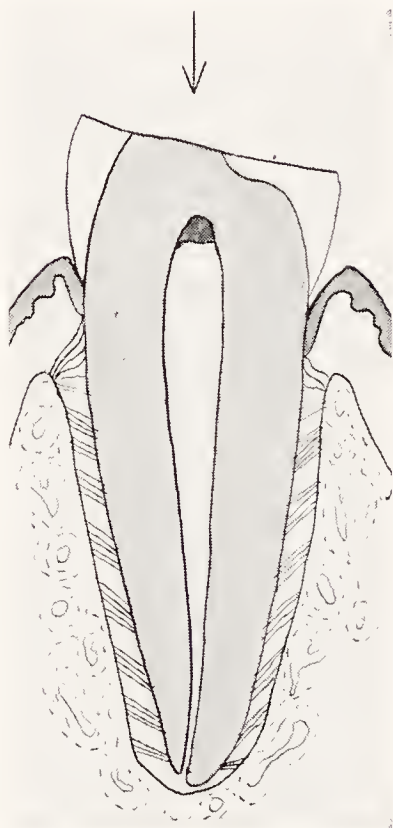


FIG. 318.—Distribution of force over the different groups of fibers in vertical (axial) occlusal stress. Lower bicuspid, with considerable occlusal wear. The arrow indicates the direction of force. All the oblique fibers are under uniform strain; only the alveolar crest fibers and some of the apical fibers are relaxed.

Vertical (Axial) Stress.—Purely vertical stress is exerted upon teeth with flat, worn occlusal surfaces if the mouth is closed in centric relation. Then the tooth is pressed into the socket parallel to its long axis. The full amount of force is taken over by the oblique fibers. All of these fibers, as well as almost all of the other fiber groups, are stretched at the same time. Inasmuch as a great deal of resistance is offered to this type of stress, the tooth will not yield except to great pressure and then only to a small extent (Fig. 318). The only fibers that are relaxed are the apical fibers, and perhaps some of the alveolar crest fibers.

Horizontal (Lateral) Stress.—Practically every stress exerted upon a tooth results in at least a certain amount of horizontal force, causing the tooth to tip. In all young intact teeth, the interlocking of the cusps in occlusion creates a horizontal force. The same is true of every lateral, grinding movement of the mandible. Stress upon the crown of a tooth is seldom purely horizontal. The closest approach to it occurs in a deep overbite in which the anterior teeth overlap entirely; in this case an attempt to thrust the mandible forward without opening the mouth results in an almost horizontal stress being exerted upon the upper and lower anterior teeth.

ANALYSIS OF THE TIPPING MOVEMENT OF TEETH. POSITION OF THE FULCRUM.

The mechanics of tipping movement will be discussed in order to explain the participation of the various groups of periodontal fibers in horizontal stress. To simplify the explanation, a single-rooted tooth with a straight, slightly tapering root will be used as an example. Case, who studied the question from the orthodontic viewpoint, compared a tooth, the crown of which is exposed to lateral (horizontal) stress, to a post in soft homogeneous soil. He describes the following experiment (Fig. 319):

“If you should drive a 4-foot post one-half its length into clayey soil of uniform quality, and then take hold of the top of the post and move it back and forth with a view of subsequently pulling it out of the ground, you would be working a lever which combines the qualities of the first and second kinds, or one like the oar in which the so-called areas of fulcrum and weight act as fulcrums to the other. After pulling the post out of the ground, if it were possible for you to make a transverse section of the soil for the purpose of examining the shape of the hole you had made, you would find it somewhat the shape of an hour-glass; the upper portion of the opening being about twice as large as the lower.” From Case’s illustration it becomes evident that the upper part of the “root” of the post moves in the same direction as the “crown” of the post, while the lower portion moves in the opposite direction. The fulcrum is below the middle of the “root”; therefore the excursion of the “apex” is less extensive than that of the upper part of the root.

Schwarz studied the question of the fulcrum experimentally and confirmed Case’s assertion. He showed that the effect of horizontal stress on a dog’s incisor is as follows: The tooth is close to the alveolar bone at the margin of the alveolus. The apex moves in a direction opposite to that of the crown and also opposite to that of the upper part of the root; however, the excursion of the apex is less extensive than that of the upper part of the root (Fig. 320). From this Schwarz concluded that the fulcrum is located at *X*, between the middle and apical third of the intra-alveolar part of the root. Further proofs, gained from animal experiments as well as from sections through human teeth, have confirmed the opinion that this location of the fulcrum is the rule in a tipping movement. In teeth with more than one root, the fulcrum is located between the roots in the bone of the interradicular septum (Fig. 328).

One reason that the apex deviates less than the root at the margin of the alveolus is the presence of the apical fibers that prevent the

apex from being displaced laterally. Johnson, Appleton, and Rittershofer have shown that in young teeth, with not yet fully-developed apices, the excursions of the root ends were very noticeable since no fibers were present to hinder apical displacement; in these teeth the fulcrum evidently was further crownward, at about the middle of the root. On the other hand, Oppenheim found that when delicate lateral stress was applied to teeth with fully developed root ends, the apex showed hardly any displacement; the amount of excursion increased steadily from the apex crownward.

In tipping movement, about one-half of the various groups of periodontal membrane fibers is stretched, and the other half is relaxed. The areas of tension and of relaxation are diagonally opposite to each other (Fig. 321). A comparison of Figure 321 with Figure 318

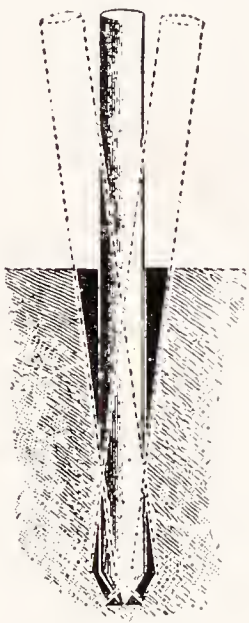


FIG. 319

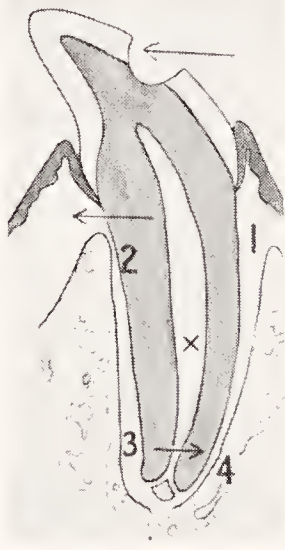


FIG. 320



FIG. 321

FIG. 319.—Case's post lever experiment: If a post sticking in a homogeneous, soft soil is moved back and forth, the fulcrum is located below the middle of the embedded part of the post. The hole in the ground, therefore, assumes the shape of an hour-glass. (Case, *Dental Orthopedia*.)

FIG. 320.—Schematic drawing of a labio-lingual section through a lower incisor of a dog. Shortly before death the tooth had been ligated to an arch wire, pulling the crown toward the lingual side. The arrow indicates the direction of this force. Notice the distribution of the width of the periodontal space around the tooth: 1, wide periodontal space at the alveolar margin on the labial side (side of pull); 2, narrow periodontal space at the alveolar margin on the lingual side (side of pressure); 3, wide periodontal space at the apex on the lingual side (side of pull); 4, narrow periodontal space at the apex on the labial side (side of pressure). The fulcrum is located near the middle of the root, at X. (Drawn after Schwarz, *Ztschr. f. Stomatol.*)

FIG. 321.—Distribution of force over the different groups of fibers in horizontal (lateral) occlusal stress. Bucco-lingual section through a lower bicuspid. The arrows indicate the direction of force. The crown is displaced toward the lingual (left) side, whereas the apex moves in the opposite direction. The fulcrum of the tipped tooth is located about in the middle of the root at X. The stretched fiber groups are indicated by heavy lines; the relaxed fibers are not included in the diagram. About one-half of the fibers on either surface of the root is exposed to a heavy strain, the other half is relaxed or compressed.

shows why teeth withstand vertical stress better than horizontal stress: in vertical stress the force is distributed evenly over almost all fibers; in horizontal stress, one-half of the fibers is stretched to the limit, the other half relaxed or even compressed.

THICKNESS OF THE PERIODONTAL MEMBRANE.

The function of the teeth has a decided influence upon the thickness of the periodontal membrane. The following observation has been made: the less function, the thinner the periodontal membrane; the more function, the thicker the periodontal membrane. Measurements of the thickness of the periodontal membrane have been reported by Coolidge, Jozat, Kellner, Klein, and Kronfeld. The smallest measurements were obtained from embedded teeth; slightly larger ones from erupted teeth without antagonists, and considerably larger measurements from teeth in intact arches and having antagonists. The thickest periodontal membranes were observed around single teeth that carried a great functional load. The following table gives some of the figures obtained by measuring the thickness of the periodontal membrane of human permanent teeth under various functional conditions.

THICKNESS OF THE HUMAN PERIODONTAL MEMBRANE (IN MM.).

Kind of tooth.	Age.	Occlusion.	Thickness of periodontal membrane at margin of alveolus.	Thickness of periodontal membrane at middle of alveolus.	Thickness of periodontal membrane at fundus of alveolus.	Average thickness of periodontal membrane.
Lower 2d bicuspid	19	Completely embedded	0.09	0.05	0.05	0.06
Lower 1st molar	45	No antagonists	0.09	0.06	0.07	0.07
Upper 3rd molar	38	No antagonists	0.10	0.06	0.06	0.09
Upper 2d bicuspid	16	Normal occlusion	0.24	0.12	0.18	0.18
Upper central incisor	16	Normal occlusion	0.32	0.12	0.24	0.23
Upper cuspid	14	Normal occlusion	0.28	0.08	0.24	0.20
Upper cuspid	24	Normal occlusion	0.24	0.15	0.16	0.19
Upper 2d bicuspid	38	Heavy functional stress; end-to-end occlusion	0.35	0.28	0.30	0.31
Upper central incisor	45	Deep overbite	0.34	0.16	0.22	0.24
Upper cuspid	47	Heavy occlusion	0.32	0.24	0.30	0.29
Upper central incisor	51	Deep overbite	0.36	0.24	0.32	0.31

From this table it is evident that the thickness of the periodontal membrane varies greatly; it is from three to four times greater in functioning teeth than in embedded ones. Gottlieb differentiated between "biological width of the periodontal space" and "physiological width of the periodontal space." The biological periodontal width is found in unerupted and embedded teeth; it represents the minimum distance between alveolar bone and cementum surface which has not been increased by function. The biological width varies from 0.06 to 0.10 mm.; it is rather uniform around all of the root.

The physiological periodontal width is found in teeth in function. As a rule, the physiological width is from two to three times the biological width. This depends, of course, upon the type and amount of functional stress exerted upon the tooth. As an average, a physiological width of 0.18 to 0.25 mm. is found. Whereas, in unerupted and impacted teeth, the periodontal membrane is uniformly thick around and along the entire root, in functioning teeth the membrane has a different thickness at different levels and may be thicker on one side of the root than on the other.

As indicated in the table, the greatest thickness of the periodontal membrane in a functioning tooth is at the margin of the alveolus, the smallest at about the middle of the root or slightly apically, and a somewhat greater thickness at the apex. This distribution of the periodontal thickness again corroborates the statement about the location of the fulcrum in a tipping tooth. Since the fulcrum is located slightly rootward from the middle of the root, the root makes its greatest excursions at the alveolar margin. At the middle of the root, near the fulcrum, an area of relative rest is found; this area makes the least excursions and has the thinnest periodontal space. Toward the apex the excursions become wider again.

By comparing teeth that have been subjected to different functional conditions, it has been possible to demonstrate that the thickness of the periodontal membrane around a root varies as a result of function. The larger the vertical component of stress, the more uniform is the periodontal thickness; the larger the horizontal component, the more do the figures obtained at the margin and at the fundus of the alveolus differ from those obtained at the middle of the alveolus. For example, the following figures were obtained from two teeth of the same jaw by measuring the periodontal width:

I. Right lower central incisor. Direction of force, almost vertical (end-to-end occlusion):

Thickness of periodontal membrane at margin of alveolus.	Thickness of periodontal membrane at middle of alveolus.	Thickness of periodontal membrane at fundus of alveolus.
0.18	0.16	0.17

II. Left lower central incisor. Direction of force, almost horizontal (deep overbite):

Thickness of periodontal membrane at margin of alveolus.	Thickness of periodontal membrane at middle of alveolus.	Thickness of periodontal membrane at fundus of alveolus.
0.26	0.10	0.24

From this comparison the relation between direction of force, excursion of the root, and thickness of the periodontal membrane is obvious. Measurements of this type can be made only on central sections, that is, on sections that run through the root canal parallel to the long axis of the tooth. If other than central sections are used, the measurements are misleading.



FIG. 322.—Comparison between the periodontal membrane of molar in heavy occlusion and molar without antagonist. Left: Periodontal membrane of molar in heavy occlusion. *C*, cementum; *PM*, periodontal membrane; *B*, alveolar bone. The periodontal membrane, which is strong and fibrous, is 0.22 mm. thick. Right: Periodontal membrane of molar without antagonist. *C*, cementum; *PM*, periodontal membrane; *B*, alveolar bone. The periodontal membrane, which is weak and atrophic, is 0.13 mm. thick. (Coolidge, Jour. Am. Dent. Assn.)

Coolidge reported measurements of the periodontal membrane of the lower right and left second molars of a man, aged fifty-four years. The lower right molar had no antagonist, whereas the left molar was in heavy occlusion. The average thickness of the periodontal membrane of the molar without antagonist is 0.10 mm., that of the molar in occlusion 0.20 mm. (Fig. 322).

Age influences the thickness of the periodontal membrane. Coolidge found the following averages: for the age group from eleven to sixteen years, 0.21 mm.; for the age group from thirty-two to fifty years, 0.18 mm.; and for the group from fifty to sixty years, 0.15 mm.

COMPARISON OF THE STRUCTURE OF THE PERIODONTAL MEMBRANE IN FUNCTIONING AND NON-FUNCTIONING TEETH.

The periodontal membrane of a tooth subjected to normal occlusal stress has strong, well-developed fiber bundles. Especially the

oblique fibers show a very dense, regular arrangement with only narrow spaces for the vessels and nerves between them (Fig. 322, left). In a non-functioning tooth only a few fiber bundles can be found; thin strands of connective tissue run through the periodontal space in different directions, sometimes parallel to the root surface (Fig. 322, right).

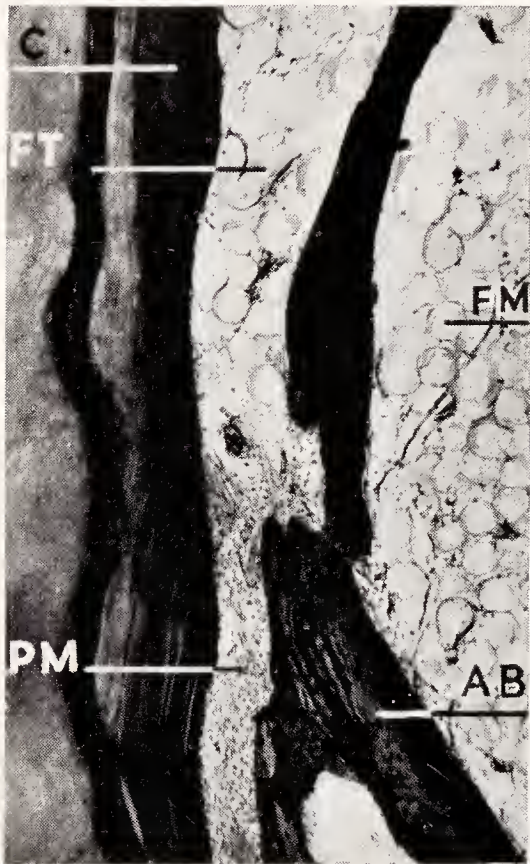


FIG. 323.—Fat tissue in the periodontal space. Upper molar without antagonist. The transformation of the fibrous periodontal membrane into fat tissue is the result of inactivity. *C*, cementum; *FT*, fat tissue between alveolar bone and root surface; *PM*, thin periodontal membrane with poorly developed fibers; *AB*, alveolar bone; *FM*, fat marrow. Thickness of periodontal membrane: 0.09 mm.

In embedded teeth the periodontal membrane is sometimes replaced by fat tissue. The same tissue is also occasionally found in the periodontal membrane of erupted teeth that have been out of occlusion for a long time. Figure 323 shows part of the root surface of an upper second molar from an old man whose mandible had been edentulous for several decades. The periodontal space is very narrow. At *FT*, the space between bone and cementum is occupied by fat tissue, the distance between bone and root being wide and irregular. Evidently in this part the periodontal membrane has entirely lost its biological and struc-

tural qualities, for there is no definite relation between tooth and bone; the tooth surface has simply been included in the system of bone and marrow spaces, and the former alveolar bone runs through the fat marrow in the same manner as the trabeculae of cancellous bone run through the jaw.

COMPARISON OF CEMENTUM IN FUNCTIONING AND NON-FUNCTIONING TEETH.

The fact that non-functioning teeth (teeth without antagonists and embedded teeth) have thicker cementum than functioning teeth of the same jaw has been known for a long time. Thick layers of cementum have been found in the bifurcations of first molars before eruption as well as on the roots of embedded cuspids. In both cases no outer stimuli could have been responsible for the deposition of cementum. Teeth without antagonists, embedded and erupting teeth seem to have an inherent tendency to move occlusally. Occlusal displacement causes widening of the periodontal space, especially at the apices, and in the bifurcation of multirooted teeth. The increased distance between root surfaces and bone is then reduced by compensatory deposits of cementum at the root ends and in the bifurcation. Kellner found that the thickness of the cementum in corresponding teeth of the same jaw was greater in the non-functioning ones; the ratio was about 3 to 2.

The arrangement and distribution of the cementum vary on functioning and non-functioning teeth. In embedded teeth the cementum is frequently arranged evenly all around the root surface, thereby transforming the root into a roundish, smooth body of cementum. In functioning teeth, however, a definite functional arrangement of the cementum is sometimes observed; spicules and circumscribed cementum hyperplasias are formed as the result of calcification at the point of insertion of Sharpey's fibers (Fig. 324). Such formations are most often found in the apical part of the root; they may be compared with the calcified attachments of tendons to bones.

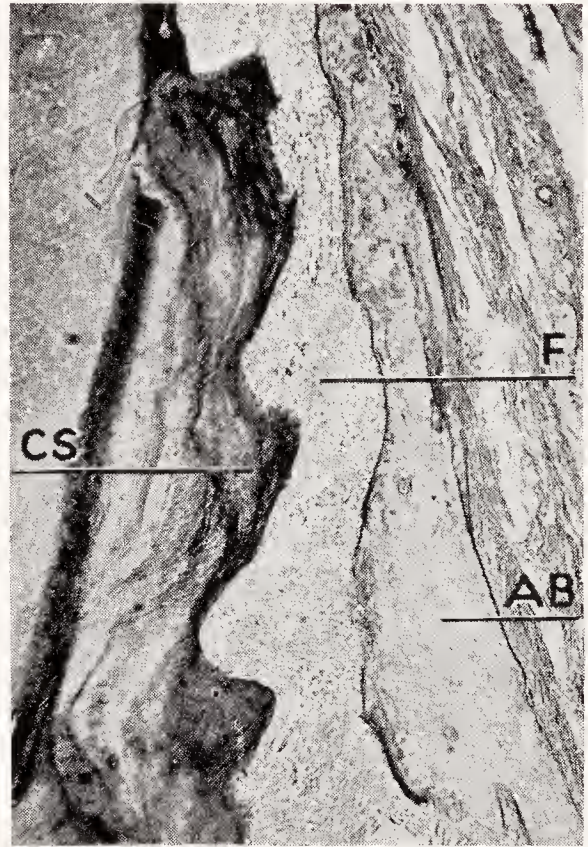


FIG. 324.—Spicules of cementum, *CS*, at the apex of a functioning tooth. *F*, fiber bundles of the periodontal membrane; *AB*, alveolar bone.

INFLUENCE OF FUNCTION UPON THE ALVEOLAR BONE.

Under physiological conditions the alveolar bone is subjected to a continuous process of tearing-down and building-up (resorption and new formation). Resorption takes place in old portions of bone

that have reached the limit of their life. This process of tissue aging is called senescence. The eliminated portions of bone are normally replaced by newly built bone. New formation is largely controlled by functional factors. Under uniform functional conditions the total mass of adult bone remains unchanged: the resorbed bone is immediately replaced by new bone, the formation of which is necessitated by the prevailing functional demand.

This balance can be disturbed in two ways, either by an increase or a decrease in function. Increased function means increased

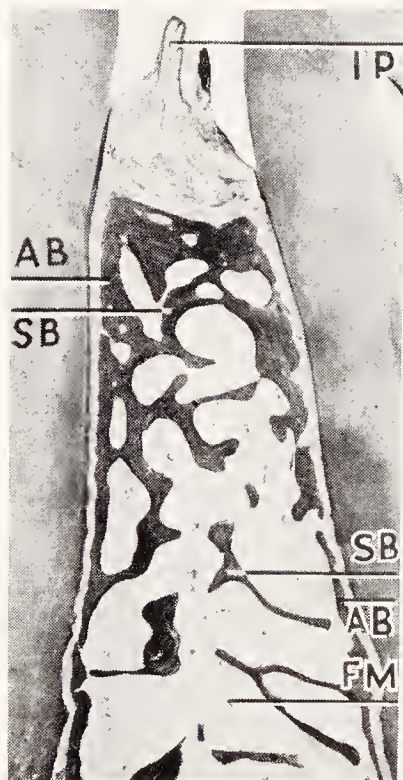


FIG. 325. — Alveolar bone, *AB*, and supporting bone, *SB*, between two lower molars. *FM*, fat marrow; *IP*, interdental papilla.

mechanical stimulus and increased activity of the osteogenetic cells; consequently, the physiological resorptive loss is overcompensated by new formation. If two units of new bone are rebuilt for each resorbed unit, the result is an increase in the total mass of bone. This increase is called functional hypertrophy. A typical example of it is the increase in the weight and thickness of the bones induced by continued heavy physical labor.

If, on the other hand, function is decreased, resorption goes on as usual; but the resorbed portions of bone are no longer replaced by an equal amount of new bone since proper functional demand is lacking. Only one unit of new bone is built for each two units that are resorbed. This leads to a reduction in the total mass of bone and results in bone atrophy (disuse atrophy, atrophy of inactivity).

Such changes are found, for example, in the bones of extremities that have been paralyzed or otherwise out of function for a long time: resorption continues, but there is no functional stimulus to cause formation of new bone, and the result is disuse atrophy of the bone.

In order to understand the functional changes in the alveolar bone it is necessary to distinguish between "alveolar bone" in the strict sense, and "supporting bone" (Orban). Alveolar bone proper consists of the outer and inner plate of the alveolus (outer surface of alveolus and wall of socket). The space between these two plates is occupied by the supporting bone, which consists of a network of trabeculae connecting the plates of alveolar bone (Fig. 325). Under changing functional conditions the alveolar bone remains rather

constant, whereas the supporting bone changes considerably. The greater the functional stress, the stronger and denser is the supporting bone. In case of lack of function the supporting bone is almost entirely missing; only the thin outer and inner alveolar plates are present (Fig. 326).

In a broad sense, the entire mandible and maxilla belong to the supporting bone. Both jaws undergo considerable change if certain functional conditions exist over a long period of time.

The difference between the structure and density of the alveolar bone around teeth under different functional conditions can some-

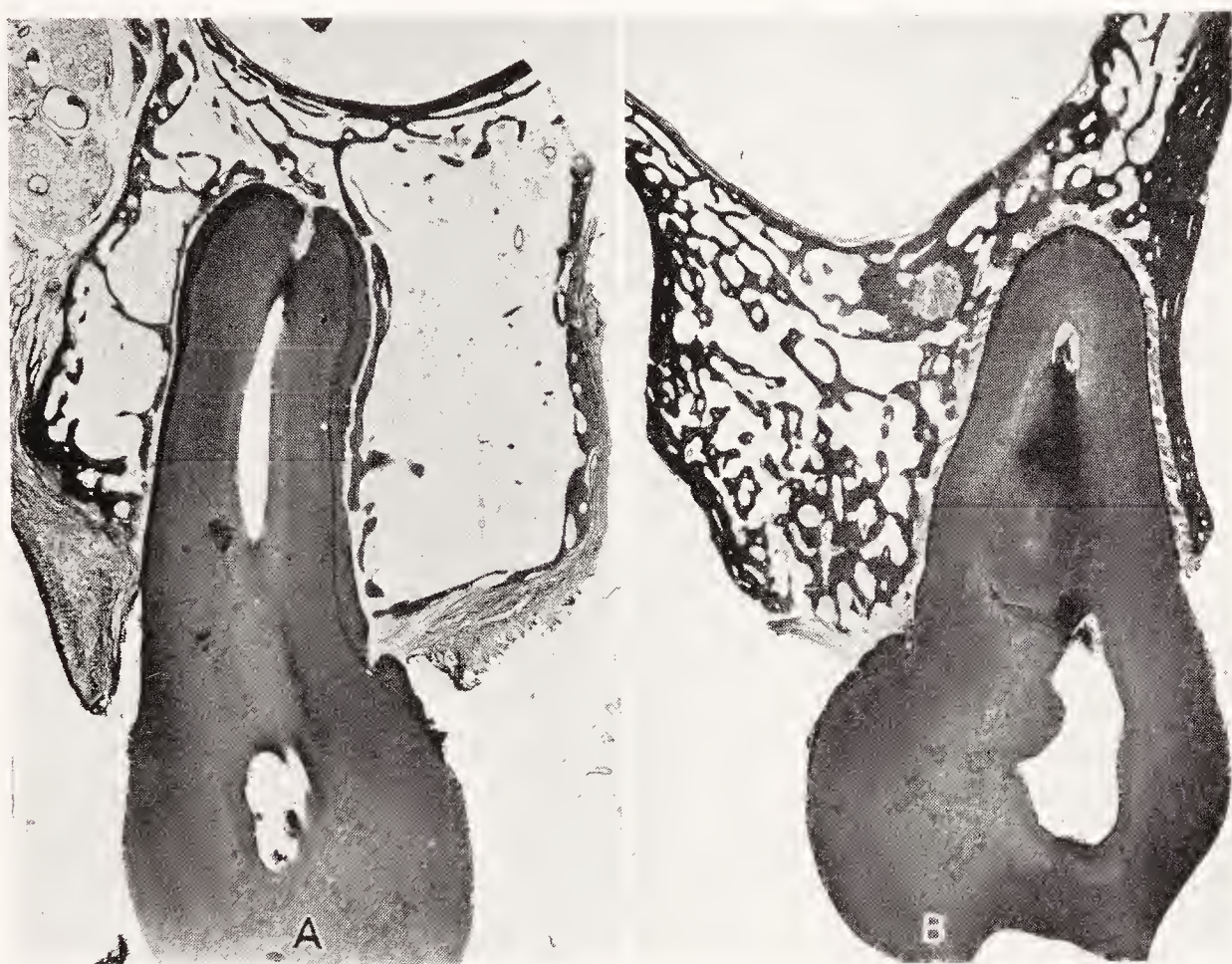


FIG. 326.—Non-functioning tooth, *A*, and functioning tooth, *B*, of the same jaw. The periodontal membrane of the non-functioning tooth is thinner and its cementum thicker than of the functioning one. There is a difference in the bone trabeculae of the jaw bone: in the non-functioning tooth the trabeculae of the spongy bone are thinner and scantier (osteoporosis), whereas in the functioning tooth the spongy bone is normal. (From Gordon, *Dental Science and Dental Art*.)

times be seen by a comparison of the radiographs. In jaws in which a number of teeth have been lost by extractions, the radiograph may reveal a thickening and condensation of bone around the teeth that are still in occlusion (functional hypertrophy of bone). Around the teeth that have lost their antagonists, the bone appears much thinner, and the structure of the supporting bone is sometimes barely visible (MacMillan). In jaws in which all teeth are present and in normal occlusion, a uniform density of bone is usually found around all teeth.

EXPERIMENTAL PRODUCTION OF VARIOUS FUNCTIONAL CONDITIONS IN THE TEETH OF ANIMALS.

In animal experiments it is possible to create certain abnormal functional conditions in the teeth. This can be done either by decreasing or increasing the normal functional stress. By grinding or extracting one of a pair of antagonistic teeth, the function can be decreased, or it can be increased by using crowns or caps that interfere with normal occlusion.

By extracting the molars of young rats on one side in either the lower or the upper jaw, Preissecker was able to compare functioning and non-functioning teeth in the same animal. He found that the thickness of the periodontal membrane of teeth without antagonists is about one-half of that of teeth in occlusion, and thus he corroborated the measurements made in human teeth under similar functional conditions.

An extensive experiment of this type was made by Gottlieb, Orban, and the author, who used dogs for their studies. The technique was as follows: Metal caps were cemented upon upper and lower molars of dogs. Some caps had oblique occlusal planes, causing a horizontal (lateral) force which resulted in a tipping movement of the teeth. Other caps had horizontal occlusal planes, creating a vertical force and causing intrusion of the teeth into their sockets. In the majority of cases the excessive force caused traumatic injuries to the periodontal tissues. These will be discussed in the next chapter; here only those findings will be described that are of importance to the mechanism of tooth movement.

An upper incisor was ligated to a labial arch wire. After forty-eight hours the crown had been displaced labially, the apex lingually; the varying width of the periodontal space at the alveolar margin and at the apex can be plainly seen (Fig. 327).

A bucco-lingual section through an upper first molar illustrates the tipping movement of a multirooted tooth (Fig. 328). As a result of lateral stress of thirty-six hour's duration, the crown moved labially, and the apices of the mesio-buccal and the mesio-lingual roots were displaced lingually. The fulcrum was located in the interradicular bone septum.

If great stress is applied over a long period of time, first the supporting bone is changed and also, finally, the configuration of the entire jaw.

The changes in the periodontal membrane in the different periods of life are: Before eruption the periodontal space around the root is narrow (biological width); few fibers are present, and the bone

does not show the dense arrangement of supporting bone. This condition may persist throughout life if the tooth fails to erupt and remains embedded in the jaw. As the tooth erupts and reaches its normal occlusion, the periodontal space becomes wider (physiological width); well-defined fiber bundles develop between root and bone; the supporting bone becomes reinforced so as to withstand

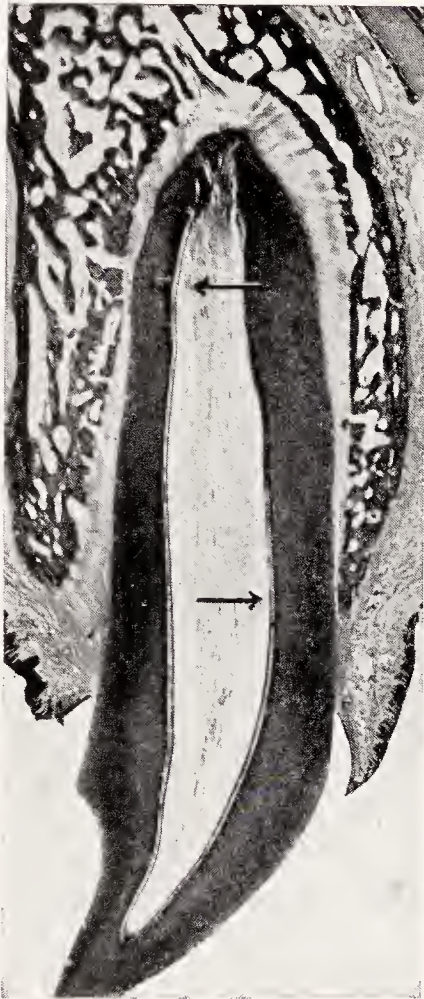


FIG. 327

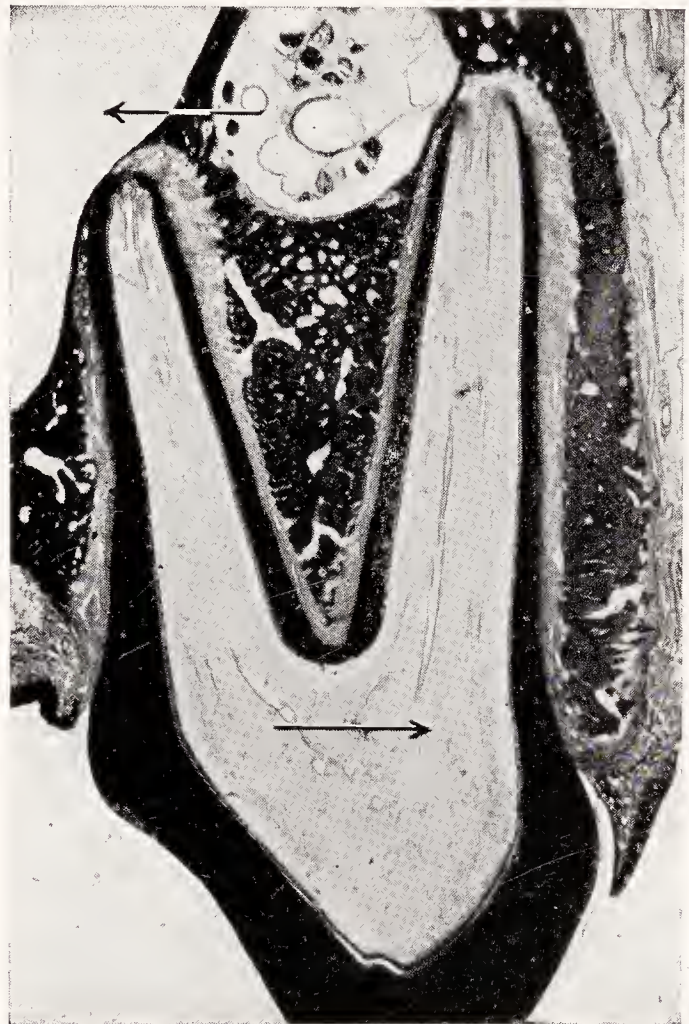


FIG. 328

FIG. 327.—Upper incisor of dog, ligated for forty-eight hours toward the labial side. The lower arrow indicates the direction of force exerted upon the crown. The apex deviated in the opposite direction. Notice the widening of the periodontal membrane in the areas of pull and the narrowing in the areas of pressure. The fulcrum is located slightly apically from the middle of the root. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

FIG. 328.—Upper molar of dog that carried metal caps with oblique planes for thirty-six hours. The lower arrow indicates the direction of movement of the crown. The apices have moved in the opposite direction. The width of the periodontal membrane is distributed accordingly. The fulcrum is located in the interradicular bone septum. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

functional stresses. Widening of the periodontal space (transformation of biological width into physiological width) is accomplished by bone resorption on the inner plate of the alveolus, caused by functional stimuli. Thus, the space between root and bone is gradually widened until the physiological width is reached.

If, on the other hand, a formerly occluding tooth is out of occlusion, either because of destruction of its own crown or loss of its antagon-

ists, the process is reversed. The periodontal membrane gradually loses its strong, fibrous structure. Its thickness is reduced by new formation of both cementum and bone, until it may finally return to its original biological width.

The physiological width permits the tooth to make excursions within physiological limits. By means of a metal separator it is possible to separate two healthy upper central incisors approximately 0.5 mm. (twice the thickness of the periodontal membrane on the distal side of each tooth) without causing the patient pain. If the separation is continued beyond this distance, the procedure becomes painful, indicating that the biological limit of the tensile strength and compressibility of the periodontal fibers has been exceeded.

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CHAPTER XV.

TISSUE CHANGES DUE TO EXCESSIVE FUNCTIONAL STRESS.

CLINICAL CONSIDERATIONS CONCERNING INCREASED OCCLUSAL STRESS.

FOR a long time the attention of dental practitioners and investigators has been directed to the possibility of injuries caused by occlusal stress.

Stillman and McCall created the term "traumatic occlusion," which they defined as abnormal occlusal stress capable of producing or having produced an injury to the periodontium. They pointed out that abnormal stress does not invariably cause injury. If one or several teeth are subjected to excessive stress, one of two things results: Either the supporting structures become reinforced, thereby making the teeth able to withstand the increased functional demand, or the supporting tissues are damaged and break down. Stillman and McCall emphasized the importance of the individual tissue reaction. A tooth subjected to "plus-occlusion" may become strengthened or weakened, depending upon the ability of the supporting tissues to adjust themselves to altered occlusal conditions.

Frequently patients are observed who have lost a number of teeth for which no artificial restorations have been made. In such mouths, single teeth bear almost the entire force of the masticatory muscles; yet these teeth are firm and solid; the health and strength of their periodontal tissues are revealed by microscopic examination. If traumatic occlusion were a primary cause for loosening of teeth, such teeth should be the first to become loose.

Another important clinical consideration is the problem of fixed bridge construction. To construct a bridge means to increase the functional load on the abutment teeth by adding the occlusal stress exerted upon the pontics. It is assumed in practice that two strong, healthy teeth or roots are able to carry the functional load of four or five teeth (their own load plus that of two or three pontics). Such bridges are successful for long periods of time, which proves that a tooth is well able to adjust itself to a considerable increase in stress.

The importance of tissue reaction is evident when, after ten or

twenty years, bridges become useless because the abutments have loosened. Frequently no changes in the occlusion can be held responsible for the loosening. Evidently for ten or twenty years the tissues around the roots of the abutments were able to adjust themselves to the increased function; but as the patient became older, the resistance of the tissues gradually diminished, and finally the roots of the abutments became unable to stand the stress.

In the study of the periodontal membrane it is difficult to draw a line between physiological and pathological conditions. Although it is usually apparent from a microscopic specimen whether the amount of function to which the tooth was subjected during life was within the physiological range, it is rarely possible to make this diagnosis in a clinical examination of the mouth. No doubt a great many of the minor traumatic injuries to which the periodontal membrane is subjected during life have no clinical effect. Therefore, all that can be done is to describe the changes produced in the tissues of human and animal jaws if the teeth have been exposed to an amount of stress evidently beyond the physiological limit; it is not possible to determine what the clinical significance of such changes is or how they can be avoided.

From the arrangement of the dento-alveolar fibers, described in the preceding chapter, it is evident that any stress exerted upon the crown is taken up by some groups of fiber bundles; some fibers are stretched, others relaxed. A physiological force may be defined as one that does not stretch or compress the fibers beyond their physiological resistance. If the force is so great that it exceeds the physiological resistance of the fibers, the root surface on the side of pressure will touch the alveolar bone, and the periodontal membrane between root and bone will be damaged, the degree of damage varying from small hemorrhages to complete crushing. On the opposite side, the side of tension, the periodontal fibers will be stretched to the limit.

Obviously the average limit of physiological force cannot be expressed in terms of pounds. The amount of stress that is physiological for one individual or one tooth may be pathological and cause damage to another.

However, several facts have been established from clinical as well as experimental observation which make the problem of pathological stress more comprehensible. One of these is that under like conditions the teeth of young people will stand more stress than those of older people. This is based upon clinical observations; it has its roots in the fact that the resistance of tissues is greatest in youth and thereafter decreases gradually with advancing age, and that

slight injuries that are readily repaired in youth may cause permanent damage in old age.

Another observation is that a tooth, under otherwise like conditions, will stand vertical stress better than lateral (horizontal) stress. The reason, as described on page 356, is that in vertical (axial) stress the force is more evenly distributed over all fibers.

The greater possibility of damage from lateral than from vertical stress has been recognized for a long time from clinical observation. Grinding of the occlusal cusps has been advocated as a prophylactic measure against loosening of the teeth. It has also been a general practice in restorative dentistry not to construct high interlocking cusps that tend to create lateral stress in mastication. The normal involution of teeth points in this direction. In people of advanced age with well-used, normal dentitions, the cusps have practically disappeared, and the occlusal surfaces are smooth, even planes. Automatically a transition takes place from the very resistant, young teeth with high cusps to the less resistant, older teeth to which lateral stress might be injurious.

From this viewpoint the practice of eliminating high cusps and lateral interlocking in older patients should be encouraged. In doing so, the dentist does what nature often fails to do in modern man, namely, to provide adequate occlusal wear.

TISSUE CHANGES CAUSED BY EXCESSIVE OCCLUSAL STRESS.

Excessive Occlusal Stress Produced Experimentally in Animals.—A force that exceeds the physiological resistance of the periodontal fibers causes injury to the periodontal soft tissues. This injury is followed, as a rule, by resorption of the bone and sometimes of the tooth also. Gottlieb and Orban cemented metal caps on molars of dogs for varying lengths of time; then teeth and surrounding bone were sectioned. Since the bite was opened by the caps, the entire masticatory force was exerted upon the crowned teeth and, consequently, caused rather extensive pathological changes. Similar changes are produced in man if a filling, crown, or bridge is placed in such a way that it interferes with the occlusion and prevents the other teeth from coming in contact.

On the side of the teeth sustaining lateral pressure, the periodontal membrane was injured to varying degrees. Thrombosis of the periodontal blood-vessels was usually the first microscopic evidence that the limit of resistance of the fiber bundles had been exceeded (Fig. 329). The periodontal membrane around the vessels shows a homogeneous structure; the nuclei and other cell elements did not

stain, which indicates that the vitality of the tissues had been lost. As a result of this tissue injury, no resorptive processes could occur in the damaged area. In order to resorb bone or cementum, the periodontal soft tissue must be vital. If the soft tissue is badly damaged, it can produce no osteoclasts; consequently no changes can take place

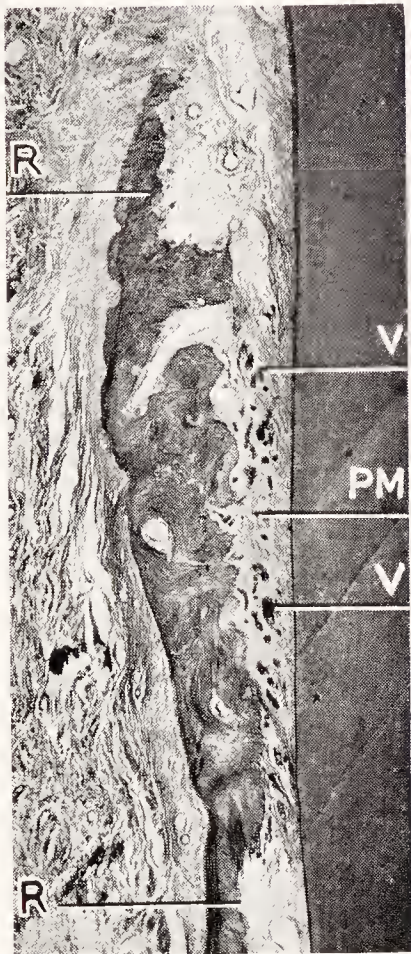


FIG. 329

FIG. 329.—Compression of the periodontal membrane and thrombosis of the blood-vessels near the alveolar margin. Dog, upper molar. *PM*, compressed periodontal membrane; *V*, thrombosed periodontal blood-vessels; *R*, bone resorption due to pressure. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

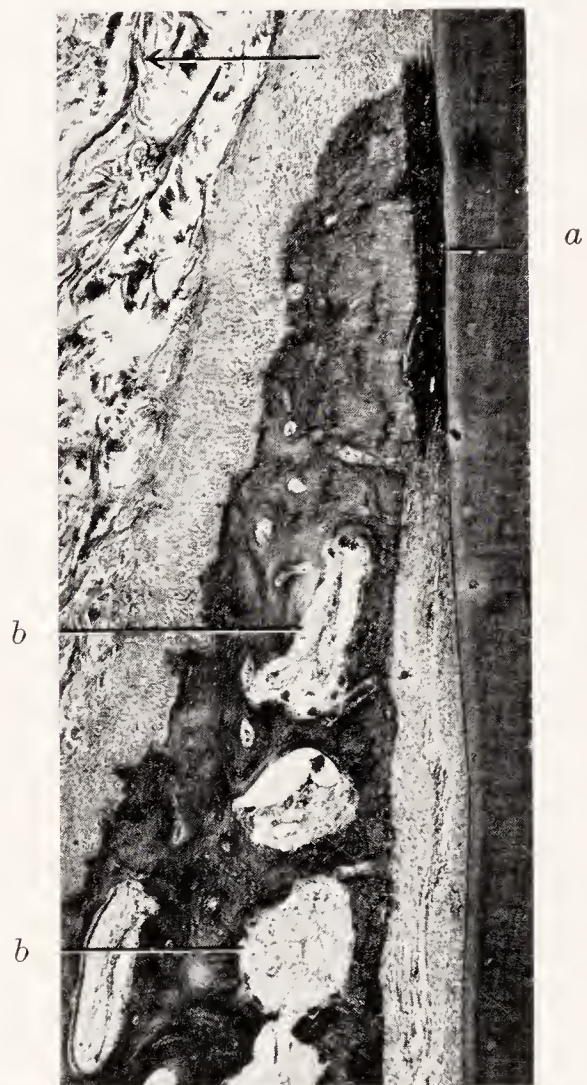


FIG. 330

FIG. 330.—Crushing of the periodontal membrane at the alveolar margin of a dog's incisor. A strong orthodontic force had been applied by means of silk ligatures for forty-eight hours. The arrow indicates the direction in which the tooth was being moved. *a*, crushed and blood-stained periodontal membrane; *b*, osteoclasts causing bone resorption in the marrow spaces. (From Gottlieb and Orban, *Die Veränderungen der Gewebe bei übermässiger Beanspruchung der Zähne*, Georg Thieme, Leipzig, 1931.)

in the two hard substances, cementum and bone. If the force is very strong, the periodontal soft tissues are completely crushed and changed into a structureless, blood-stained mass (Fig. 330). If this condition is allowed to persist for some time, the dead tissue between cementum and bone is worn away. Then no further displacement of the tooth in the direction of pressure is possible.

After a necrotic space between bone and tooth has once developed, bone resorption starts at a distance from the center of tissue injury. Osteoclasts are found in the marrow spaces of the alveolar process and in that part of the periodontal membrane in which normal cell structure is still present. From these points, undermining resorption occurs toward the bone adjacent to the necrotic periodontal membrane until this bone has been completely undermined and gives way for further movement of the tooth. In this way the tooth may finally reach a position in which it is no longer subjected to excessive stress.

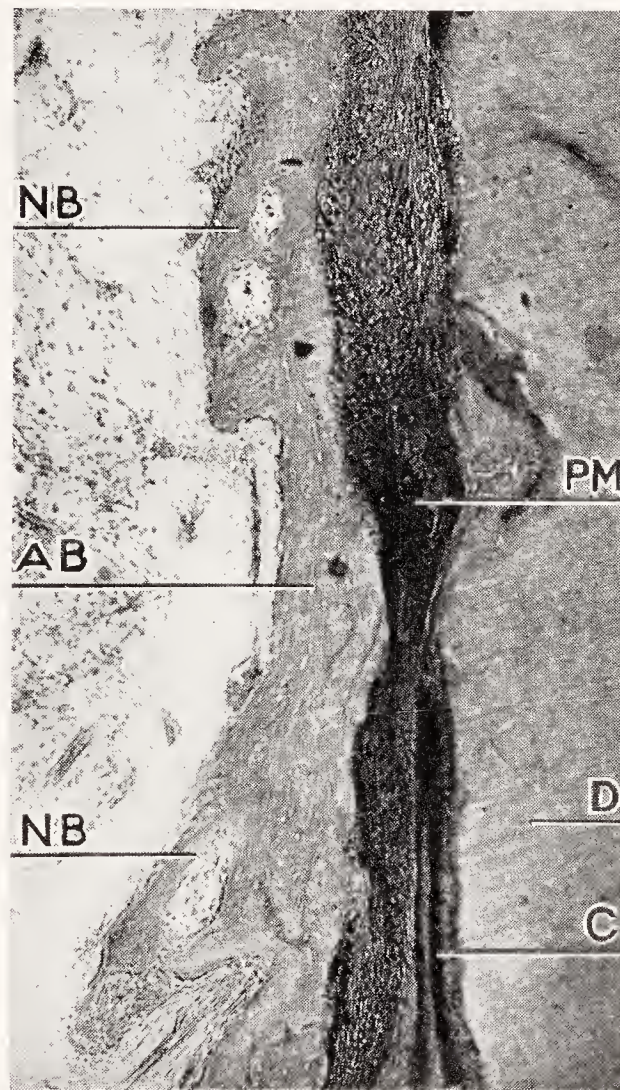


FIG. 331.—Compressed blood-stained periodontal membrane on the mesial root surface of a human lower molar that was tipped mesially. *D*, dentin; *C*, cementum; *PM*, compressed periodontal membrane; *AB*, alveolar bone; *NB*, compensatory formation of new bone on the side of the bone away from the tooth. (Orban, Jour. Am. Dent. Assn.)

Changes in Human Periodontal Tissue Caused by Excessive Occlusal Stress.—A large number of animal experiments has established certain tissue changes as typical under given conditions of stress. There might be some doubt as to the applicability of these findings to man were it not for the fact that histological investigations on human jaws with teeth subjected to excessive stress

showed exactly the same type of pathological changes. Such findings have been reported by Bauer, Coolidge, Grohs, Häupl, Lundquist, Orban, and others. Figure 331 shows an area of necrotic, blood-stained periodontal tissue between the inner alveolar plate of bone and the mesial side of the root of a lower second molar. The tooth had been tipped mesially following the loss of the first molar. The excessive tipping force resulted in a crushing of the periodontal membrane on the side of pressure. A similar but more extensive destruction of the periodontal soft tissues is seen in Figure 332, which was taken of the bifurcation of another lower molar in the same jaw. A necrotic space was formed between bone and root surface, which was accompanied by resorption of both bone and tooth and by a

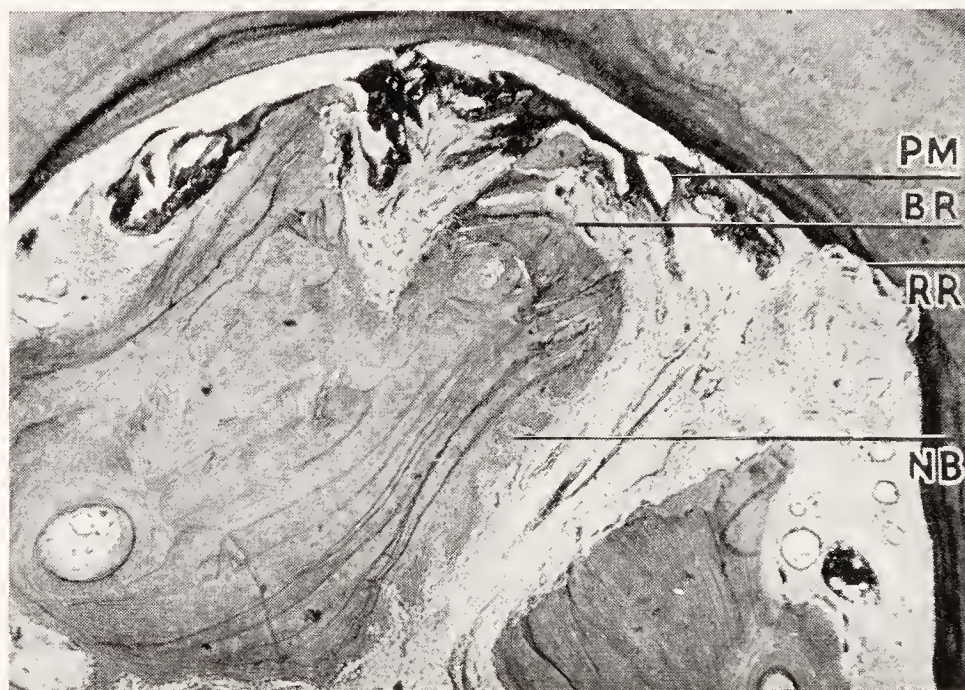


FIG. 332.—Crushing of periodontal membrane between root surface and bone. Human lower molar, bifurcation. *PM*, crushed and blood-stained periodontal membrane; *BR*, undermining bone resorption; *RR*, root resorption; *NB*, formation of new bone on the side of the bone away from the tooth. (Orban, Jour. Am. Dent. Assn.)

reparative new formation of bone in areas more distant from the point of destruction. The dark masses in the periphery of the necrotic space are remnants of old hemorrhages. The bone adjacent to these areas was undermined by resorption; finally the entire bone that originally bordered the crushed periodontal membrane was eliminated, and the tooth was again able to move in the direction of force. In Figure 333 the final outcome of undermining resorption is illustrated: The periodontal space is very wide and is filled with tissue debris and coagulated blood; a large number of giant cells are working on the final elimination of these tissue remnants. At the same time the bone adjacent to the newly established periodontal space is being resorbed.

The great number and extent of the necrotic areas in the periodontal membrane in this particular case make it probable that the poor general health and lowered resistance of the patient, who died of tuberculosis after a long illness, may have had something to do with this condition. It is probable that the occlusal forces which formerly had not exceeded the tensile strength of the periodontal fibers may have become traumatic and caused injuries

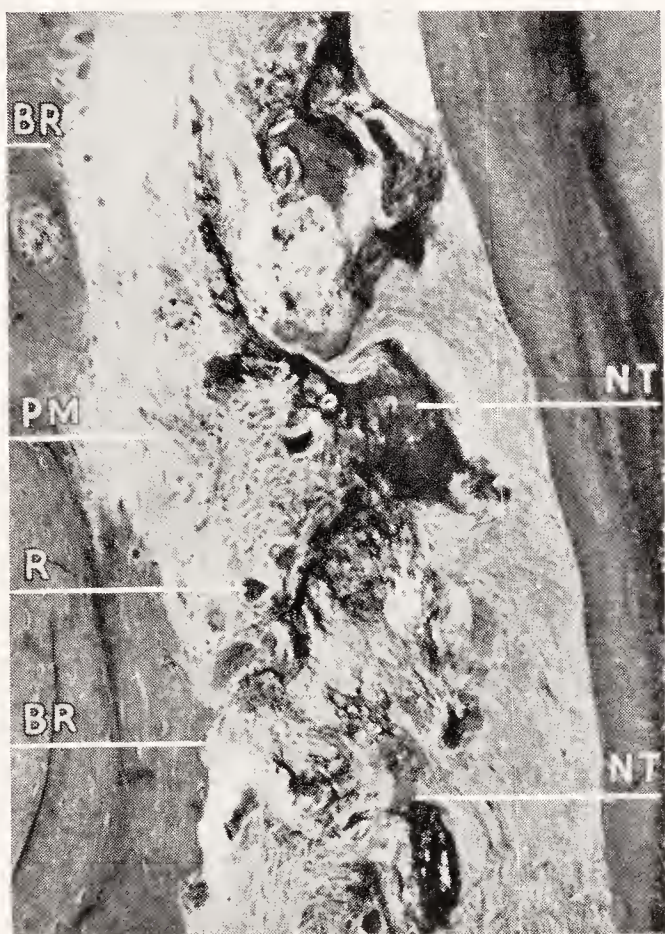


FIG. 333.—Final outcome of undermining resorption: resorption of the necrotic periodontal membrane (human specimen). *NT*, compressed necrotic connective tissue of the periodontal membrane; *R*, giant cells resorbing the tissue debris; *PM*, partly regenerated periodontal membrane; *BR*, resorption of alveolar bone. (Orban, Jour. Am. Dent. Assn.)

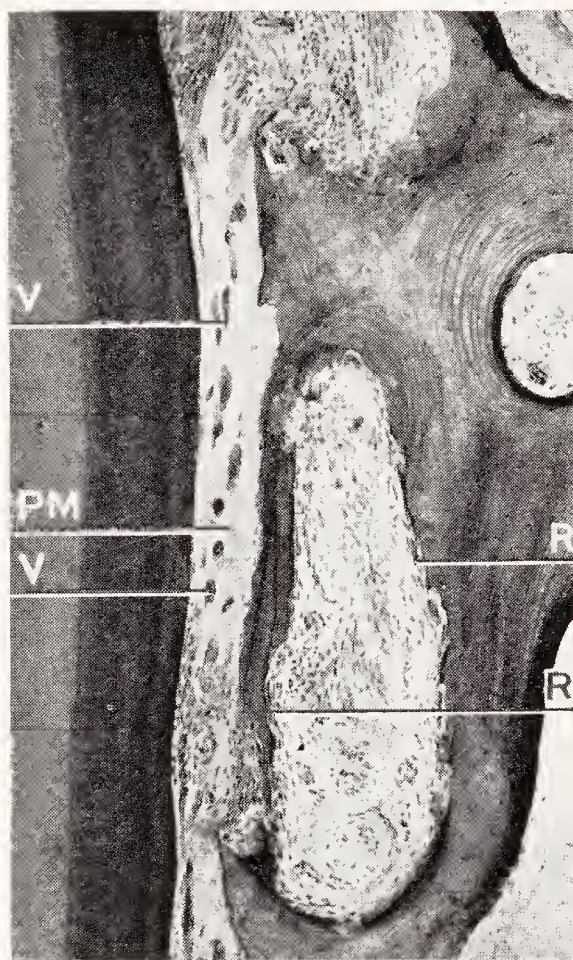


FIG. 334.—Thrombosis of the vessels of the periodontal membrane caused by occlusal trauma. Human lower incisor. *PM*, compressed periodontal membrane; loss of cell structure; *V*, thrombosed vessels; *R*, undermining resorption in the marrow spaces. Compare with Figure 329. (Grohs, Ztschr. f. Stomatol., courtesy of Urban and Schwarzenberg, Berlin-Wien.)

to the tissues after the general resistance of the body was lowered by the severe illness.

A similar case was reported by Grohs. In the jaws of a man, aged forty-eight years, a great number of teeth had been lost by extraction, leaving an excessive amount of occlusal stress on the remaining teeth. The periodontal tissue of a lower lateral incisor had been crushed between the root and the bone near the alveolar margin by an excessive tipping force exerted upon the crown

(Fig. 334). The blood-vessels of the periodontal membrane became thrombosed, and the surrounding tissue was transformed into a homogeneous, structureless mass. Bone resorption took place in the marrow spaces behind the necrotic periodontal membrane, eventually completely undermining and eliminating the bone.

If excessive stress is exerted upon a tooth in a vertical (axial) direction, the periodontal membrane may be crushed at the fundus of the alveolus, causing contact between root end and bone at this point. Such a condition is illustrated in Figure 335. It shows the root tip of a human upper molar that was exposed principally to

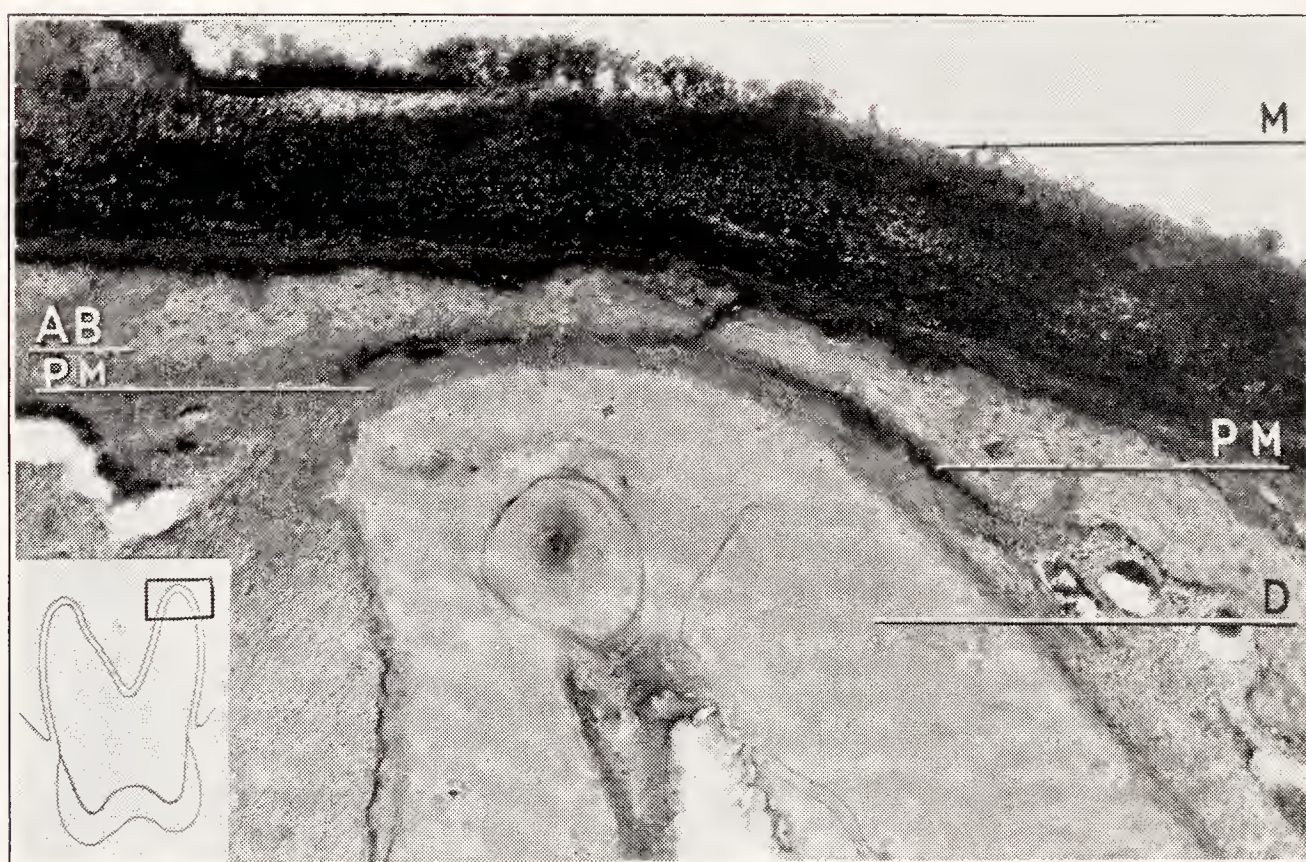


FIG. 335.—Apex of lingual root of an upper first permanent molar. The insert in the lower left corner indicates the location of the area shown in the photomicrograph. *D*, dentin; *AB*, alveolar bone; *PM*, periodontal membrane showing necrosis from excessive occlusal stress; *M*, floor of maxillary sinus. (Coolidge, Jour. Am. Dent. Assn.)

vertical stress. At the apex the periodontal membrane has been crushed, and the cementum surface is in contact with the bone.

Tissue changes like the ones illustrated in Figures 331 to 335 cause few clinical symptoms except perhaps a slight soreness of the tooth. Such changes occur very frequently; almost all human jaws that have been examined microscopically show some minor traumatic injuries of this kind. However, these injuries usually have no clinical significance and do not interfere with the retention of the tooth, unless the individual resistance is unusually low.

The etiology of such traumatic injuries may be illustrated by a

practical example: A dentist sets an inlay that is slightly too high and dismisses the patient without checking the occlusion. The tooth with the inlay, at least in some positions of the jaws, will have to bear the entire force of mastication. A few days later the tooth is usually sensitive to pressure, and sometimes it is slightly loose. If the stress upon the tooth is relieved by grinding the inlay to normal occlusion, these symptoms disappear in a day or two.

From the knowledge gained from animal experiments as well as from human jaw material, the following tissue changes can be expected in such a case: Immediately following the placing of the inlay (or any other restoration that interferes with the occlusion) the periodontal membrane is compressed in some areas, and minute hemorrhages occur in it. The tissue is destroyed on the side of pressure, and the root comes in contact with the bone. These changes are manifested by a slight soreness of the tooth (traumatic pericementitis).

If at this stage the force exerted upon the tooth is relieved by grinding, the tooth assumes its former, normal position in the socket; the necrotic tissues are resorbed, and a normal periodontal membrane is regenerated.

If the normal occlusion is not restored, the necrotic areas are undermined from the periphery by bone resorption until the root is able to move in the direction of pressure and until the tooth has assumed a position in which the occlusal force is normal. Then reparative changes take place, reestablishing normal function in the new position. If, however, tissue resistance and vitality are low, the traumatic tissue injuries are not repaired; on the contrary, larger areas of the periodontal membrane become involved until extensive destruction and resorption of the alveolar bone may lead to the loss of the tooth.

The studies of human and animal tissues corroborate what Stillman and McCall call "potential" and "actual" traumatic occlusion. Every increased force involves the potentiality of occlusal trauma, as indicated by microscopic injuries that do not show clinically; but only if the reparative power of the organism fails does actual traumatic occlusion develop and the tooth become permanently damaged.

Slightly increased occlusal stress does not cause necrotic changes in the periodontal membrane. If a filling is only a little too high, perhaps not more than enough to stretch some fiber groups more than others, the vitality of the periodontal membrane will not be damaged; bone resorption takes place in the areas of pressure, relieving the

periodontal soft tissues, and the force is neutralized by a minute shift in the position of the tooth.

In this connection an experience encountered in the practice of orthodontia might be mentioned. When an excessive amount of force is applied to a tooth in orthodontic movement, the tooth sometimes moves rapidly at first, but then comes to a standstill. It is then necessary to relieve the tooth of all force for a few weeks before carefully starting the movement again. What has happened in this case? The excessive force stretched the fibers beyond their tensile resistance and crushed the periodontal membrane between root and socket on the side of the pressure (Fig. 330). Because of this injury the periodontal membrane could not produce osteoclasts, so that any further orthodontic movement was made impossible for the time being. After the tooth had been released, the necrotic membrane was gradually resorbed and replaced by normal tissue; and not until the periodontal tissue had been completely repaired and was able again to produce osteoclasts was further orthodontic movement possible.

Functional Stress and Root Resorption.—The findings in experimental occlusal trauma have shown that the bone is much more easily resorbed than the tooth. If a tooth is pressed against the socket, the bone, as a rule, is eliminated; the tooth, however, remains intact or is only slightly resorbed. Resorptions due to occlusal trauma found in the microscopic examination of human teeth are usually small and have little clinical significance (Fig. 336). Moreover, such shallow resorptions of the root surface are repaired as soon as the trauma subsides. Practically all human teeth that have been moved orthodontically and later examined microscopically show shallow resorptions, most of which are already repaired (see Chapter XVI). In dogs in whom root resorptions had been produced by experimental occlusal trauma, reparative changes took place if, after the caps were removed, the animal was kept alive for a sufficient length of time to allow cementum deposition on the resorbed dentin surface (Fig. 337).

As a whole, root resorption due to occlusal trauma is insignificant as compared to idiopathic root resorption of systemic origin, which causes extensive destruction of teeth. In the entire problem of root resorption the constitutional factor seems to be of far greater importance than external influences upon the teeth (trauma, inflammation). In individuals with high resistance, even a great amount of occlusal trauma does not produce resorption of the roots. On the other hand, in patients susceptible to root resorption, normal mastication or

careful orthodontic treatment may occasionally produce progressive root resorption, leading finally to complete destruction of the root.

Functional Stress and Gingival Crevice.—The influence of occlusal stress upon the attachment of the gingival tissues is still being debated. In the teeth of dogs, no gingival changes were produced even when great forces were applied. In an incisor of a sheep Box found an increase in the depth of the crevice after a metal crown had been worn for one hundred and four days.

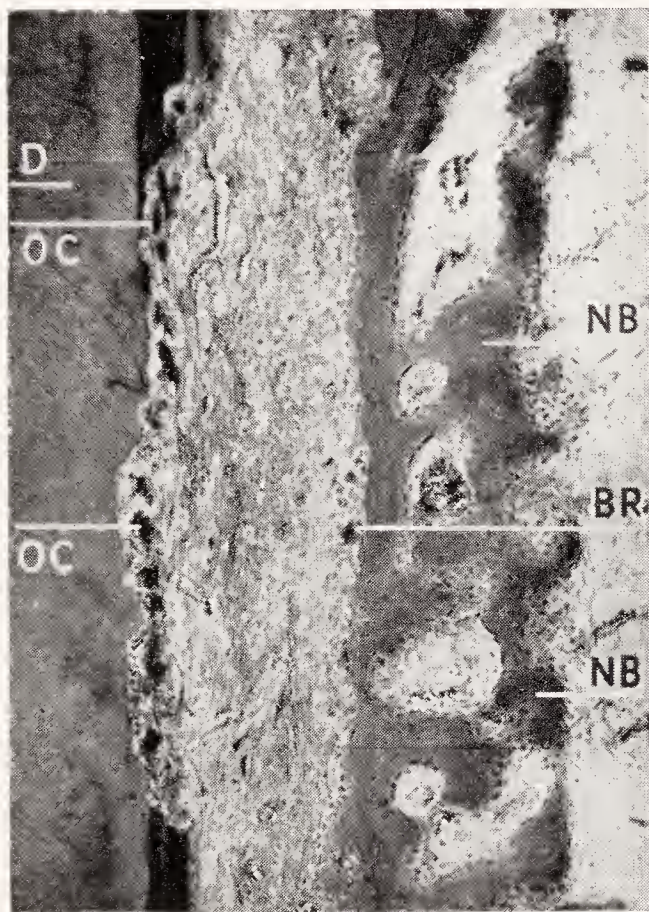


FIG. 336



FIG. 337

FIG. 336.—Root resorption due to excessive occlusal stress. Human tooth. *D*, dentin; *OC*, osteoclasts resorbing cementum and dentin; *BR*, bone resorption; *NB*, formation of new bone on the outer side of the alveolus. (Orban, Jour. Am. Dent. Assn.)

FIG. 337.—Repaired root resorption on a dog's molar. The resorption was produced experimentally by placing a metal cap on the tooth for nearly six months; then the cap was removed and the animal kept alive for an additional six months. *D*, dentin; *C*, original cementum surface; *C'*, cementum deposited upon resorbed dentin surface after removal of caps; *PM*, fiber bundles of periodontal membrane regenerated in the resorbed area; *AB*, alveolar bone projecting into the resorbed area (functional repair, see Figure 250). (From the experiment of Gottlieb and Orban.)

The findings in human teeth subjected to heavy occlusal stress vary. In individuals with high resistance there may be no pathological changes in the gingivæ; in others, recession of the gingivæ and formation of pockets are observed. This is particularly true of horizontal force. Then the injury to the alveolar margin on the side of pressure may cause a detachment of the gingival soft tissues

and the formation of a deep pocket in this area. Such a condition has been illustrated in Figures 301 and 302.

Functional Stress and Pulp Changes.—Intensive use of a tooth causes wear and subsequent reduction in the size of the pulp chamber by secondary dentin formation (see page 68). Denticles (pulp stones) are frequently found in the abraded teeth of older people; they have no significance other than that they are the expression of the tendency toward pulp calcification and reduction in the size of the pulp chamber.

It appears improbable that a pulp could die merely because of excessive functional stress. The bundle of blood-vessels at the apical foramen seems to have enough tensile strength and elasticity to adjust itself to the excursions of the apex and to keep the pulp vital. However, mild forms of pulp irritation (hyperemia) are occasionally observed in overloaded teeth and are relieved by balancing the occlusion.

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CHAPTER XVI.

TISSUE CHANGES IN ORTHODONTIA.

ORTHODONTIC therapy brings about changes in the position of the teeth and in the form of the jaws and face. By applying different kinds of forces to the teeth and jaws, the position of the teeth, the structure and arrangement of the surrounding bone, and the relative position of the upper and lower arches are influenced and altered.

The tissue changes in orthodontia are of interest both to the practitioner and to the scientific investigator. A tooth is normally anchored firmly in its alveolus and yields only fractions of a millimeter even if a relatively great force is applied to it for a short period of time. Still, it is known from orthodontic practice that delicate forces, if applied over a period of months or years, are able to cause marked changes in the position of the teeth and in the bone. This observation aroused the interest of the early investigators, and two theories were offered to explain the bone changes in orthodontic tooth movement.

Kingsley and Walkhoff¹ explained the orthodontic movements of the teeth as the result of compression and extension (elasticity) of the alveolar bone. If a tooth were forced by an appliance to move in a certain direction, they assumed that the bone is compressed on the side toward which the tooth is moving and extended or stretched on the opposite side of the root.

The other theory, offered originally by Schwalbe and Flouren,² was based on the assumption that the alveolar bone is resorbed and eliminated by osteoclastic activity on the side of pressure, while new bone is built on the opposite side, the side of tension.

FINDINGS IN ANIMAL EXPERIMENTS.

In order to study the tissue changes in teeth subjected to orthodontic forces, Sandstedt³ placed appliances on the incisors of a dog and moved the teeth toward the labial or lingual side. He then prepared sections through the jaws, which showed osteoclasts and bone resorption on the side of pressure, and osteoblasts and bone

¹ Cited by Oppenheim.

² Ibid.

³ Ibid.

formation on the side of tension. Oppenheim performed similar, more extensive experiments on the incisor teeth of a young monkey, using arch wires anchored to the molars. The incisors were attached to the arch wire by ligatures, and simple forms of tooth movement, such as tipping, elongation, and depression (shortening) were performed. After forty days the animal was killed, and the teeth and jaws were sectioned and studied microscopically.

By means of these specimens Oppenheim demonstrated that the position of the teeth in the jaw can be changed only by means of resorption and new formation of the surrounding bone. Wherever pull is exerted, new bone is formed, arranged in the direction of the pulling force, and wherever pressure is exerted upon the alveolus, the bone is resorbed, thus creating space for the intended movement of the tooth. Oppenheim's findings definitely disproved the theory of elasticity of bone.

Besides the resorption and apposition of the bone surface directly facing the root, there is also a general rearrangement of the alveolar bone around the moved tooth. The formerly solid and even bone plate of the alveolus is transformed into spicules of bone that are arranged parallel to the direction of force; each spicule shows resorption on one end and new formation of bone on the other. The tearing-down and building-up of bone on each spicule continues as long as the orthodontic force is applied to the tooth. If orthodontic movement is discontinued and the tooth is retained in its new position, the arrangement of the alveolar bone gradually changes until the original solid bone plate is again restored.

A few examples will serve to illustrate the fundamental bone changes obtained by orthodontic treatment in monkeys.

Tipping of a Tooth.—In a labio-lingual section through an incisor that has been tipped labially or lingually, a distinction must be made between the side of pressure, toward which the tooth moves, and side of tension, where pull is exerted upon the alveolar bone. In labial movement, for instance, the lingual surface of the alveolus is the side of pull. On that side new spicules of bone are built, arranged parallel to the direction of the stretched periodontal fibers (Fig. 338). The end of each spicule that faces the tooth is densely beset with osteoblasts, whereas the end that is farthest from the tooth shows osteoclastic resorption. In lingual movement, a similar arrangement of the bone is found on the labial side, the side of pull. There is a tendency for new bone to fill in behind the moving tooth on the side of tension. When the orthodontic force is first applied, the periodontal membrane fibers on the side of pull are stretched

and the periodontal space is widened. As a result, bone formation is stimulated on the inner wall of the alveolus, which tends to reduce the periodontal space to its normal width. This process continues as long as force is exerted upon the crown.

On the side of pressure the bone is also transformed into spicules arranged parallel to the direction of force; but the distribution of

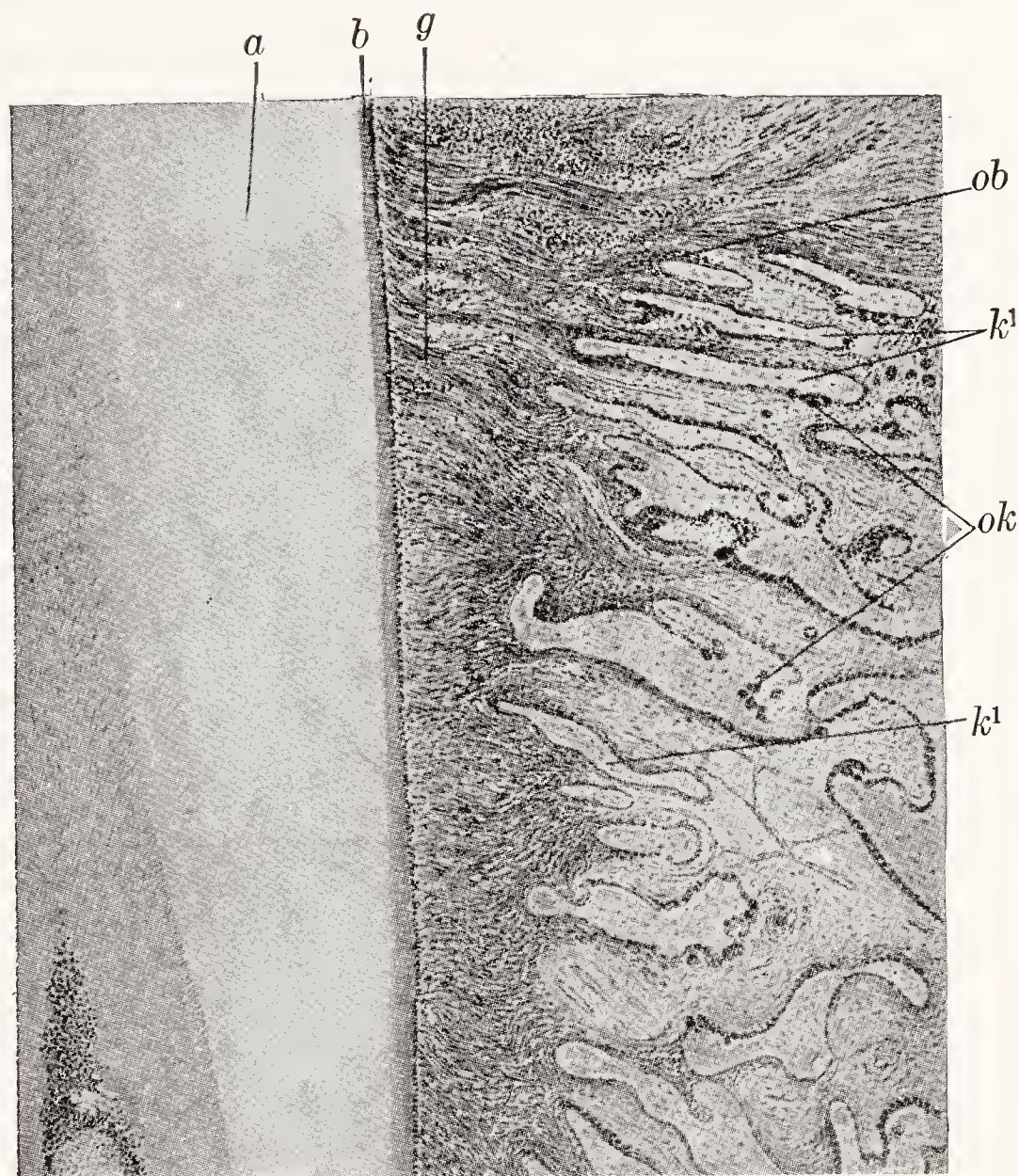


FIG. 338.—Labial movement, lingual side. Monkey incisor moved orthodontically for a period of forty days. Near the alveolar margin the bone is arranged in long spicules, k^1 , oriented at right angles to the long axis of the tooth. Each spicule is densely beset with osteoblasts, ob , on the end facing the tooth; at the opposite end, the bone is being resorbed by osteoclasts, ok . a , dentin; b , cementum; g , periodontal membrane. (Oppenheim.)

resorption and new formation on each spicule is exactly opposite to that found on the side of pull. Each spicule shows resorption on the extremity next to the tooth surface and formation of bone on the end away from the moving tooth (Fig. 339).

The biological significance of these changes is plain. The labial and buccal plate of bone covering the roots are very thin; therefore, in labial or buccal movement, the resorption of the bony plate on

the side of pressure would rapidly lead to complete destruction of the labial or buccal bone were it not for simultaneous formation of new bone. An outer plate of bone is constantly maintained, which gradually moves more and more toward the buccal or labial side as the tooth advances in that direction.

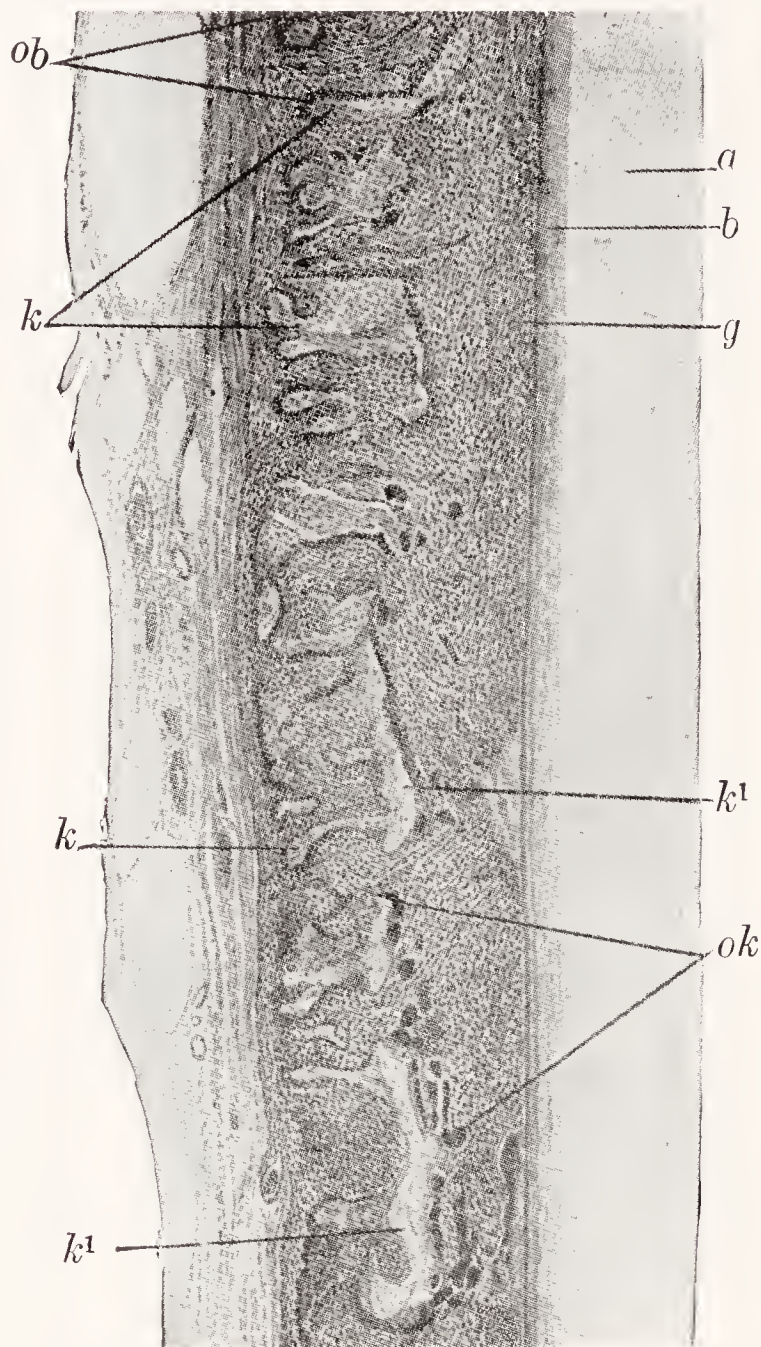


FIG. 339.—Labial movement, labial side. Incisor of monkey. Near the alveolar margin the compact bone has disappeared and has been replaced by spongy bone, *k*, with orientation of the spicules at right angles to the long axis of the tooth. At the end that faces the tooth, each spicule is beset with osteoclasts, *ok*, and at the opposite end with osteoblasts, *ob*. *k*¹, remains of the original bone; *a*, dentin; *b*, cementum; *g*, periodontal membrane. (Oppenheim.)

Bone resorption on the inner side of the alveolus on the side of pressure is the result of a decrease in the space between tooth and bone. When the orthodontic appliances are put in place and force is applied, the tooth is forced against one side of the socket, and pressure is exerted upon the intervening periodontal membrane. As a result, the periodontal membrane is stimulated to osteoclastic

activity; the bone is resorbed, and the pressure upon the soft tissues is relieved. Bone resorption enables the tooth to move in the direction indicated by the orthodontic force; this movement continues until the force has expired.

In tipping movement, if gentle forces are used, the bone changes are greatest near the alveolar margin and from there steadily decrease toward the apex. This would indicate that the fulcrum of the tipping tooth is located at the apex. In the discussion of the influence of function upon the teeth, it was stated that the fulcrum of a tipping tooth is located in the apical third of the root, so that the apex moves in a direction opposite to the rest of the root (see page 357). Both of these apparently contradictory statements hold true, although under different conditions: Powerful tipping forces will cause the apex to move opposite to the direction of the crown, while gentle tipping forces will have so little effect that excursions of the apex may be disregarded. Hence with delicate forces, as are applied in modern orthodontia, the fulcrum is located at or near the apex; with strong forces the fulcrum is located between the middle and the apical third of the root.

Johnson, Appleton, and Rittershofer carried out experiments similar to those of Oppenheim and reported similar results. They worked on upper central incisors of young monkeys. In their specimens the apex shows a marked deviation toward the side opposite the direction of movement of the crown. A possible explanation of the difference between the findings of Oppenheim (no displacement of the apex) and those of Johnson, Appleton, and Rittershofer (apex displaced in a direction opposite to that of the crown) may be this: in Oppenheim's experiment the roots were fully formed and apical fibers were present that anchored the root tip to the fundus of the alveolus, whereas in the experiment of the American investigators the apical foramina were still wide open and no apical fibers had as yet developed; therefore, the apices were more easily displaced by tipping forces.

In this connection the question arises: If gentle pressure is applied to the periodontal membrane, why is only the bone resorbed and not the tooth surface as well, since the amount of pressure exerted upon the tooth surface is identical to that exerted upon the bone? Apparently there is a biological difference between tooth and bone surface. Cementum is, under otherwise identical conditions, much more resistant to resorption than bone, and the difference is the basis for the practice of orthodontia. Were this not so, were cementum and bone equally subject to resorption, the tooth would be

resorbed just as much as the surrounding bone, which would make orthodontic therapy impossible.

Elongation of a Tooth.—The elongation of a tooth results in the formation of new bone at the fundus of the alveolus and at the alveolar margin. At the fundus of the alveolus, the bone spicules are arranged in the direction of the pull, parallel to the long axis of the



FIG. 340.—Elongation. Apex of the tooth. Incisor of monkey. Bone spicules, k^1 , arranged in the direction of pull. The upper end of each spicule is surrounded by osteoblasts, *ob*, the lower end by osteoclasts, *ok*. *a*, apex. (Oppenheim.)

tooth (Fig. 340). Osteoblasts are present on each spicule at the end toward the tooth, and osteoclasts at the opposite end. At the alveolar margin, formation of new bone can be found along the entire circumference of the elongated tooth (Fig. 341); in this way the alveolus evidently adjusts itself to the new, raised position of the tooth.

Depression of a Tooth.—In orthodontic depression (shortening) of a tooth, the bone changes are exactly opposite to those found in elongation. There is resorption of bone on the entire inner surface of the socket, but especially at the fundus of the alveolus and at the alveolar margin. Apparently the alveolus tends to adapt itself to the new position of the tooth.

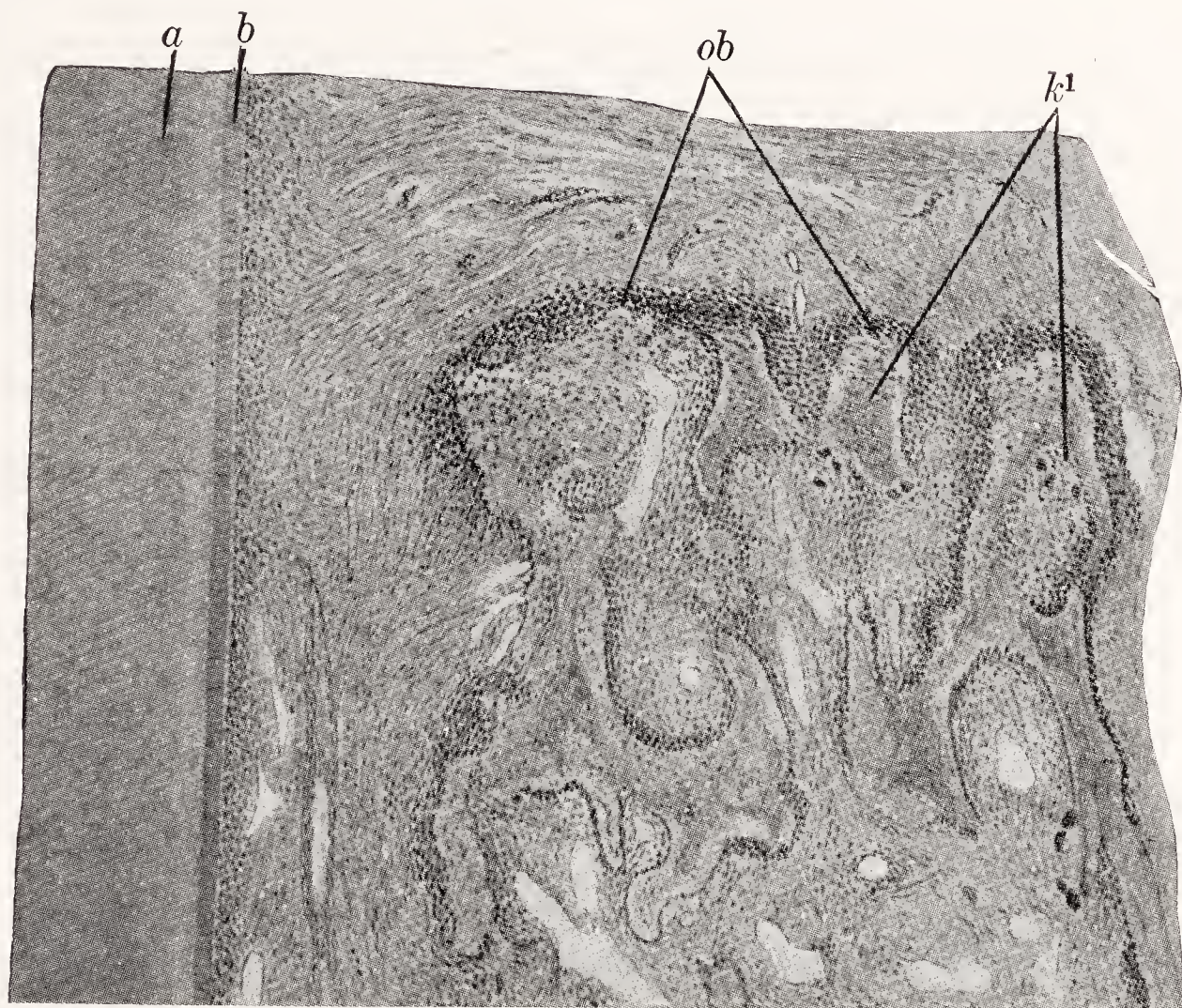


FIG. 341.—Elongation. Lingual alveolar margin. Newly formed spicules, k^1 , of spongy bone arranged in the direction of the pull. The spicules show osteoblasts, ob , at the upper end and osteoclasts at the lower end. a , dentin; b , cementum. (Oppenheim.)

Tissue Changes During the Period of Retention.—Oppenheim showed that after the active orthodontic force has been discontinued and the tooth is retained in its new position, characteristic changes take place in the alveolar bone. During the time of active treatment the bone is arranged in spicules parallel to the direction of force. During the period of retention these spicules are gradually transformed back into the solid alveolar bone plates. New Haversian systems develop, and the bone assumes the character of lamellated bone. The final arrangement of the bone and the form of the alveolus depend largely upon the prevailing anatomical conditions and upon the functional requirements that the tooth has to meet in its new position.

BONE CHANGES AROUND HUMAN TEETH FOLLOWING ORTHODONTIC MOVEMENT.

For a long time the findings in animal experiments were the only source of information about orthodontic tissue changes. And as long as corresponding human specimens were not available, these

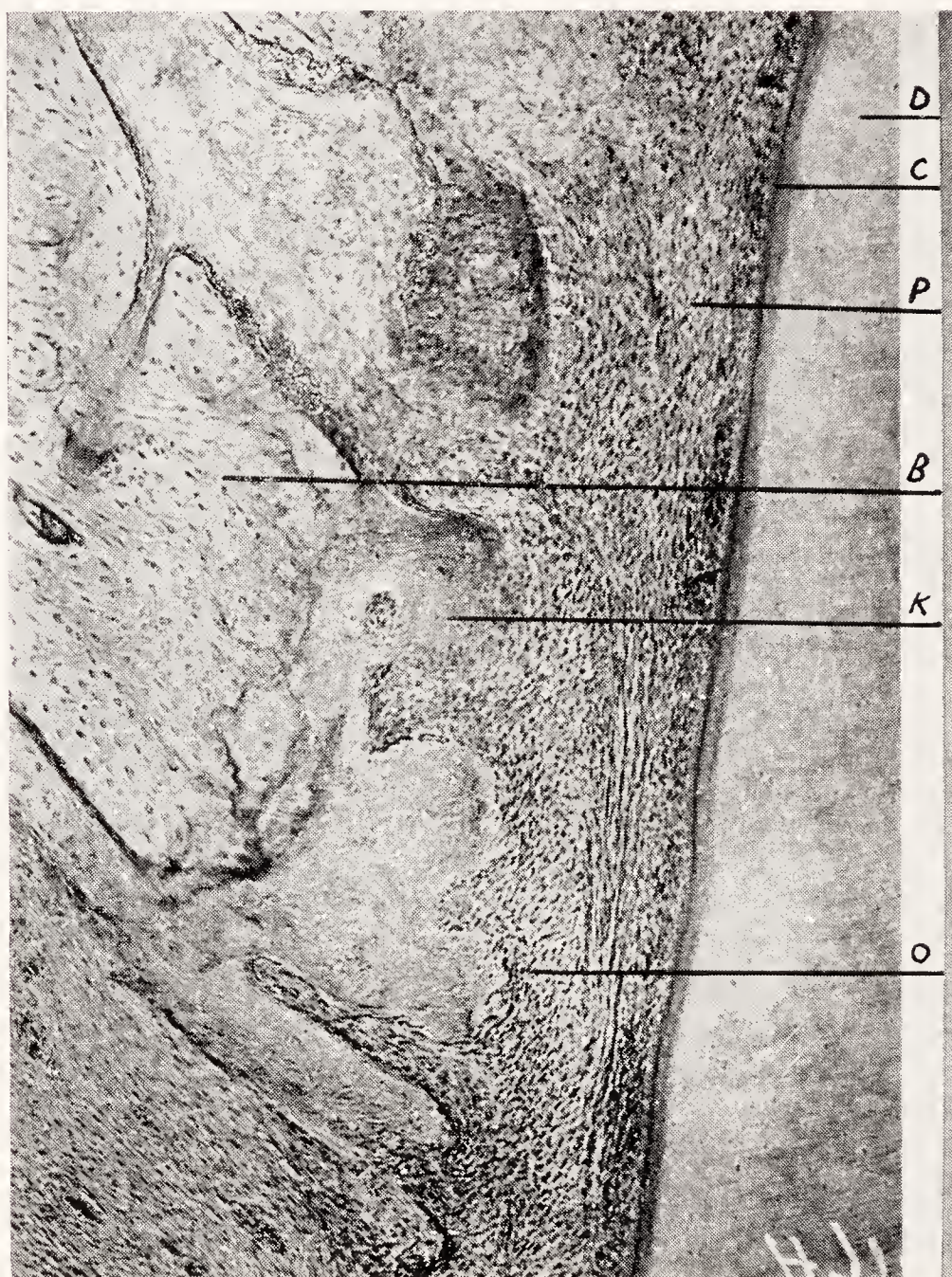


FIG. 342.—Upper first bicuspid of child, aged thirteen years. The tooth was moved labially by an auxiliary spring for twenty-nine days. The illustration shows the lingual bone crest (side of tension). *D*, dentin; *C*, cementum; *P*, periodontal membrane; *O*, osteoblast; *K*, new bone; *B*, old bone. (Stuteville, Angle Orth.)

findings were applicable to man only with reservations. The first human tooth that was moved orthodontically and then studied microscopically, together with part of the surrounding tissues, was reported by Herzberg. Its microscopic examination revealed changes very similar to those produced by Oppenheim in monkeys under identical conditions. The bone spicules were arranged in the

direction of pull at right angles to the surface of the tooth. On the side of pull each spicule of bone was lined by osteoblasts and osteoid at one end, whereas bone was being resorbed at the other end.

More extensive investigations of this kind have been carried out during the last few years by Oppenheim and by Stuteville. Accurately gauged forces were used, and good specimens were obtained



FIG. 343.—Buccal bone crest (side of pressure) from the same tooth as that shown in Figure 342. Resorption of bone. *D*, dentin; *C*, cementum; *P*, periodontal membrane; *L*, osteoclast; *B*, old bone. (Stuteville, Angle Orth.)

by biopsy (Figs. 342 and 343). The teeth used for this purpose were usually upper first bicusps that had to be extracted in order to correct a malocclusion. The microscopic examination of two-rooted teeth indicated clearly from the distribution of pressure and tension that the apices had deviated in a direction opposite to that of the crown (Fig. 344). The location of the fulcrum in tipping

movement depends on several factors, such as intensity and direction of the force and shape and number of the roots. But, as a rule, there is some deviation of the apex, although this deviation is always less marked than the displacement of the cervical portion of the root (see Fig. 328).

QUANTITATIVE EVALUATION OF ORTHODONTIC FORCES.

In a series of experiments performed on dogs, Schwarz approached the problem of determining the relationship between an orthodontic force of known intensity and the tissue changes produced by this force. He used an appliance that consisted of a strong arch wire anchored to the molars and canines. This arch wire lay on the lingual side of the premolar teeth. Auxiliary springs were soldered to it in such a way that the premolars were moved buccally. The force exerted by these springs had been previously measured so that each premolar was subjected to a force of known intensity.

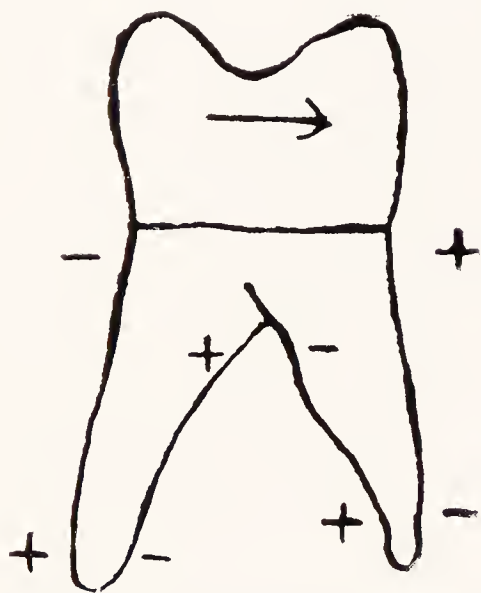


FIG. 344.—If a tooth with two roots is tipped, four areas of pressure (+) and four areas of tension (—) develop. (Oppenheim, Angle Orth.)

The appliances were left in place for a period of several weeks; then the animal was killed, and the teeth together with the surrounding jaw bone were sectioned. The microscopic ex-

amination showed a definite relationship between the amount of the force and the type of tissue reaction. Schwarz divided the orthodontic forces into four groups according to their biological reaction:

First Degree of Biological Reaction.—The force is of such short duration or so gentle that it causes no definite reaction in the periodontal tissues.

Second Degree of Biological Reaction.—The force is gentle in a biological sense: Schwarz assumes that it does not exceed the capillary blood pressure, and produces a continuous resorptive process on the side of pressure. The result is the desired orthodontic movement without damage to the tissues.

Third Degree of Biological Reaction.—The force is fairly strong: it is slightly in excess of the capillary blood pressure in the periodontal membrane on the side of pressure, causing anemia and injury

to the soft tissues. No bone resorption can occur in the damaged area, but usually after the damaged areas have been removed by undermining resorption, the tissues regenerate; however, this kind of force is likely to cause root resorption.

Fourth Degree of Biological Reaction.—The force is strong: it crushes the periodontal membrane on the side of pressure, bringing the root surface into contact with the bone. There is even a possibility of the pulp dying because of the tearing and hemorrhage at the apex. Later, extensive bone resorption takes place, with the probability of root resorption.

Stuteville applied known orthodontic forces to human teeth, which were later examined microscopically, and concluded that the experimental findings of Schwarz cannot be directly applied to man. He observed no root resorption in teeth that had been moved with forces of 150 to 200 gm.; yet, in other teeth root resorption occurred after the application of much weaker forces. Aside from individual variation, it is not so much the amount of force that determines the tissue reaction as the distance over which the force is active.

RESULTS OF EXCESSIVE FORCES IN ORTHODONTIA.

Injury to the Periodontal Membrane.—The periodontal membrane consists of soft tissue situated between cementum and bone. If an excessive force is applied to the tooth, this soft tissue is compressed and its blood supply reduced or completely cut off. As a result, the blood-vessels are thrombosed, and the tissues become asphyxiated and eventually necrotic (Fig. 345). Such areas of tissue necrosis between cementum and bone on the side of pressure have been observed by all investigators who have applied strong orthodontic forces to human or animal teeth.

Following this kind of injury to the periodontal membrane, no changes are at first visible in the vicinity of the damaged tissues. This explains the clinical observation that intense force used in orthodontia will sometimes lead to the complete standstill of a tooth rather than to the expected movement. An intact periodontal membrane is essential to bone resorption. If the periodontal membrane is damaged to such an extent that no osteoclasts can be produced, bone resorption cannot take place, and, consequently, orthodontic movement is impossible.

The work of Oppenheim and other investigators introduced biological thinking into the practice of orthodontia. Now it is

comprehensible why the use of intense forces does not lead to better or faster results than the use of gentle forces. It is necessary to give the tissues time to adjust themselves to the changed condition: the bone must be resorbed and rearranged, and new bone must be built to make up for that which is lost by resorption. If too much



FIG. 345.—Labial alveolar crest of an upper second incisor of a young dog. The tooth was moved labially by an auxiliary spring over a period of fourteen days. The periodontal membrane was squeezed between tooth and bone and became necrotic. *D*, dentin; *C*, cementum; *P*, periodontal membrane; *N*, area of necrosis of the periodontal membrane; *R*, undermining resorption; *B*, bone. (Stuteville, *Int. Jour. Orth. Oral Surg.*)

force is applied, the periodontal soft tissues are damaged, and no coördinated orthodontic movement is possible until these tissues have completely recovered.

Root Resorption Associated With Orthodontic Treatment.—In order to understand the problem of root resorption in orthodontia,

a distinction must be made between microscopic root resorption and extensive destruction of the root.

Microscopic Areas of Root Resorption.—During the past ten years many human teeth that had been subjected to orthodontic treatment have been examined histologically. Without exception these



FIG. 346.—Upper first bicuspid of a child of fourteen years. The tooth was moved labially by means of ligatures for eighty-two days and then held in the new position for twenty-eight days. Repaired resorption on the root surface. *D*, dentin; *C*, cementum; *T*, tooth resorption; *S*, secondary cementum; *P*, periodontal membrane; *K*, new bone; *B*, old bone; *O*, osteoblast. (Stuteville, Angle Orth.)

teeth showed shallow areas of resorption on the side of pressure. Sometimes only the cementum was affected; more often the resorption extended into the superficial layers of the dentin. These resorptions occurred regardless of type of appliance, intensity of force, and duration of treatment. They were observed by investigators in the United States (Herzberg, Stuteville), in Europe

(Grubrich, Gubler, Oppenheim), and in Japan (Kogure, *et al.*). Apparently *any* force exerted upon a tooth that is strong enough to move it is also strong enough to cause minute injuries to the periodontal tissues and to cause shallow root resorptions (Fig. 346). The only kind of force that might be expected not to cause root resorption would be one resembling the eruptive force or the force involved in spontaneous drifting of teeth; but such a force would be so weak and therefore would act so slowly that it would be worthless in orthodontic practice.

The most important fact in connection with this type of root resorption is that the resorbed areas are repaired by deposition of secondary cementum as soon as the pressure is removed. This statement is based upon the findings of Oppenheim and Stuteville, and upon clinical experience. Neither tooth nor periodontal membrane suffers any lasting damage, and only the histologist can detect the formerly resorbed area, which is now a healed scar on the root surface.

Extensive Root Resorption.—The publications of Ketcham, as well as the use of routine radiographic examination, have revealed the occurrence of extensive root resorptions in young patients. This destructive process usually starts at the apex, which in the radiograph appears as if cut off with a sharp instrument; in more advanced cases the apical one-half or even two-thirds of the root may be missing, and the tooth may be markedly loosened. Usually new bone is formed at the same rate at which the root is destroyed, so that a fairly uniform periodontal space is maintained around the remaining portion of the root. The pulp retains its vitality.

The etiology of this kind of root resorption was unknown for a long time. Ketcham suggested that certain types of orthodontic appliances were responsible. Marshall made an experimental study of root resorption. He found that animals kept on a deficient diet were much more likely to develop root resorptions from orthodontic treatment than control animals on a balanced diet. However, there was no indication of a corresponding dietary deficiency in young people with extensive root resorption. Moreover, extensive root resorption was observed in individuals who had never undergone orthodontic treatment. For this condition the terms “idiopathic root resorption” or “genuine root resorption” have been suggested. Recently Becks made a clinical and laboratory study of 100 patients with root resorption, most of them children or young adults. Fifty of them had been treated by orthodontists, and 50 had not. A thorough medical examination of all of these patients

was made, with special regard to the function of the endocrine glands, and it was found that the most prevalent disturbance was

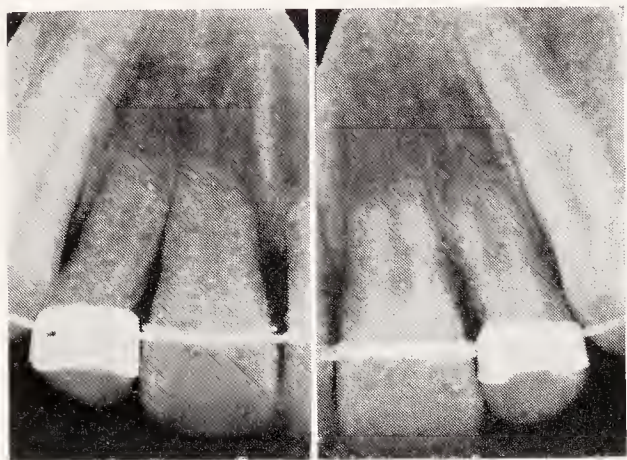


FIG. 347

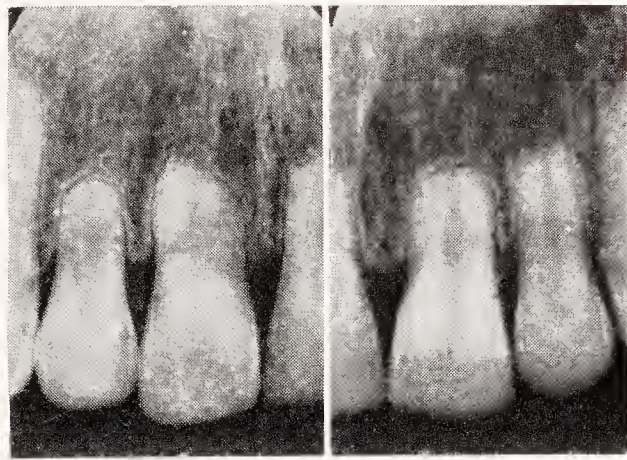


FIG. 348

FIG. 347-351.—Root resorptions. (Becks, *Int. Jour. Orth. Oral Surg.*)

FIG. 347.—Root resorptions following orthodontic treatment for three years. Age, fifteen years. Systemic disease: hypothyroidism.

FIG. 348.—Advanced root resorptions of anterior and posterior teeth following four years of orthodontic treatment. Apical periodontal membrane space has again reached normal width, suggesting arrest of the resorptive process. Note extreme osteoporotic lesion over apices. Age, thirty years. Systemic disease: hypothyroidism.

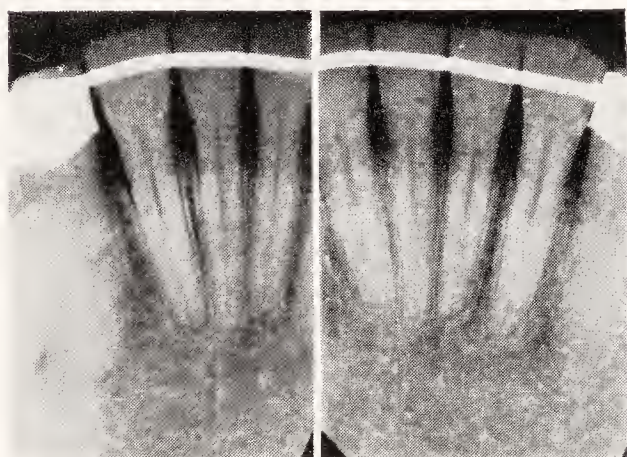


FIG. 349

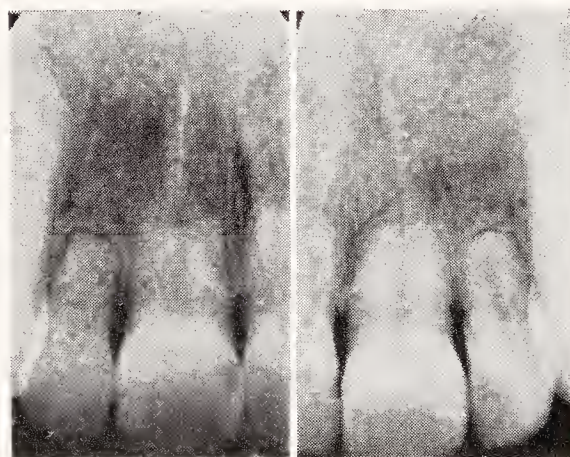


FIG. 350

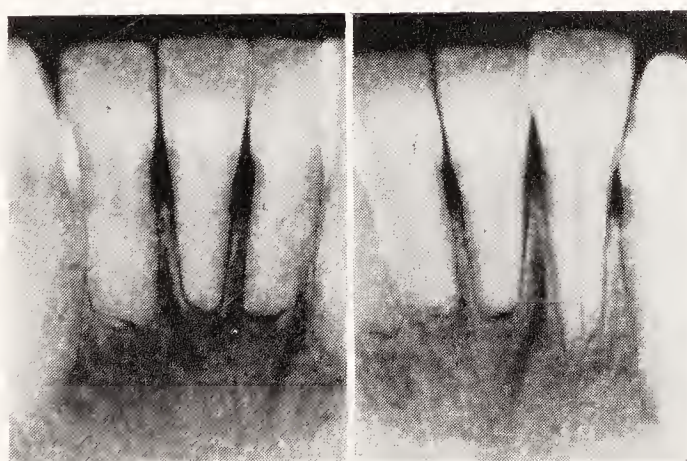


FIG. 351

FIG. 349.—Resorption of roots of mandibular anterior teeth accompanied by osteoporotic lesion and slight horizontal atrophy. Age, thirty years.

FIGS. 350 and 351.—Marked resorptions in maxilla and mandible following orthodontic treatment for seven years. Enlarged circulatory channels in mandibular anterior region with pathological bone formation. Age, thirty-six years. Systemic disease: hypothyroidism.

an underfunction of the thyroid gland (hypothyroidism). Other forms of endocrine dysfunction were observed less frequently. Orthodontic treatment apparently acted as a contributing factor but could not be considered the primary cause (Figs. 347-351). No definite correlation could be established between root resorption and the type of orthodontic treatment or appliance.³

The outcome of idiopathic root resorption is uncertain. If the resorptive process comes to a standstill before more than about one-third of the root has been lost, the tooth usually remains firm and continues to function normally. If more than one-half of the root is destroyed the prognosis is doubtful, since the tooth is likely to loosen and eventually to be lost.

The problem of root resorption is by no means solved. The work of Becks is a great step forward, but it will require much additional work and further collaboration between dentist, internist, and endocrinologist before the final answer can be given. Case reports, however, like the one recently made by Carman, are encouraging. The patient, a girl aged fourteen, was developing extensive root resorption during orthodontic treatment. A careful physical examination was made, and the diagnosis was hypothyroidism, hypocalcemia, and hypoglycemia. Orthodontic treatment was discontinued for a year, during which time a carefully planned treatment of the systemic disturbance was carried out. After this, orthodontic treatment was resumed and completed without evidence of additional root resorption.

To the practitioner the possibility of root resorption should be an added incentive for the use of radiographs in routine mouth examinations. Thus, many cases of idiopathic root resorption can be detected early, and although at present no generally applicable method of arresting the destructive process is known, at least special precautions can be taken with all dental operations, especially with orthodontic treatment.

Changes in the Shape of the Root Following Orthodontic Treatment.—Radiographs of teeth subjected to orthodontic treatment in early life sometimes show blunted, shortened roots, different from other roots in the same mouth. The history of these teeth suggests that some force may have deformed the developing root ends. Johnson, Appleton, and Rittershofer found that Hertwig's sheath at the open foramen of a young, moved tooth showed a distinct deformity that was not present in a control tooth of the same animal. In the experiments of Gottlieb and Orban, root deformities were produced by applying orthodontic forces to very young teeth. In

these teeth Hertwig's sheath appeared deformed and folded. If an animal were kept alive long enough to allow the apex to form completely, a corresponding deformity was found in the root end. The entire root was shorter and blunter than the control teeth of the same animal.

These findings in experimental animals have not yet been corroborated by similar microscopic findings in man; still they suggest such possibilities for human teeth. Blunted or shortened root ends following orthodontic movement may be due either to resorption of the apex or to a deformity and distortion of Hertwig's sheath; the latter can be suspected if the tooth in question was moved in early life before root formation was completed. Only by microscopic examination can it be decided which one of the two processes took place.

INFLUENCE OF ORTHODONTIC MOVEMENT OF DECIDUOUS TEETH UPON THE PERMANENT TOOTH GERMS.

Breitner and Tischler experimentally studied the question of whether or not orthodontic movement of deciduous teeth has any influence upon the position of the underlying tooth germs of the permanent teeth. This work was carried out on young monkeys. The microscopic examination of the bone surrounding the germs of the permanent teeth revealed that the latter always followed the movements of the deciduous teeth.

Those permanent tooth germs that were located between the roots of the corresponding deciduous teeth simply followed the movement of the latter. The permanent tooth germs toward which the deciduous roots were being moved advanced in the same direction as the deciduous tooth; and if the deciduous tooth was moved away from the permanent tooth germ, the latter followed.

These observations are an additional argument in favor of early orthodontic therapy. They justify the expectation that treatment of malocclusion in the deciduous dentition may have a beneficial effect upon the alignment of the permanent teeth.

CHANGES IN THE MESIO-DISTAL RELATIONS OF THE JAWS.

It is possible to change the mesio-distal relationship between upper and lower jaw by the use of intermaxillary rubber bands, inclined planes, and head caps. Breitner reported experimentally produced changes in the relative position of the jaws of monkeys.

He used *en bloc* anchorage, covering several posterior teeth in each jaw with caps to which intermaxillary rubber bands were fastened by means of hooks. In one case he moved the whole lower jaw forward, as it is done for the correction of a Class II occlusion (Angle) in man; but since the animal had normal occlusion to begin with, he created a Class III (Angle) malocclusion. In another animal Breitner reversed the appliances and moved the mandible from its normal position (Class I) into a Class II relation. A third experiment, "jumping the bite," was carried out by cementing metal caps with oblique planes on the posterior teeth.

The histological changes found when a mandible was moved forward or backward by means of intermaxillary rubber ligatures can be divided into three groups:

Movement of all teeth that were used for anchorage and upon which the force acted.

Changes in the form of the mandible (lengthening or shortening) produced by rearrangement of the bone of the ramus, the angle, and the condyloid process.

Displacement of the mandible at the base of the skull by changes in the mandibular fossa.

Mesial Movement of the Mandible.—After the mandible had been moved forward over a period of eighty-two days by means of orthodontic rubber bands, the animal, which by this time showed a marked Class III malocclusion, was killed. In the mandibular fossa formation of new bone was found on the distal wall; the bone was arranged in horizontal spicules, indicating the presence of a forward pulling force at the condyle (Fig. 352). On the mesial wall of the mandibular fossa, the bone was resorbed, indicating an incipient mesial displacement of the joint and of the entire mandible. New bone was found on the distal side of the ramus and the condyle, and there was resorption at the angle of the mandible.

These findings indicate that the mandible had become longer (addition of bone at the posterior side of the ramus) and its angle more obtuse (resorption at the angle).

Distal Movement of the Mandible.—The appliance used was the same as in mesial movement except that the hooks were arranged in the opposite direction and pulled the mandible backward. The distribution of the bone changes in the mandibular fossa was exactly opposite to that in case of mesial movement; there was resorption on the distal wall of the fossa and formation of new bone on the mesial wall. Thus, the mandibular fossa seemed to be adjusting itself to the intended distal displacement of the condyle.

The ramus and the condyloid process showed resorption on the distal surface, while new bone was being formed at the angle of the mandible. Such a distribution of bone changes tends to make the mandible shorter, and the angle between the ramus and body sharper.

"Jumping the Bite."—Caps with oblique planes were cemented on the posterior teeth for forty-six days; the oblique planes caused a forward sliding of the mandible during the closing movement of the jaws. In the joint, changes similar to those in mesial movement were found, corresponding to a forward displacement of the condyle.

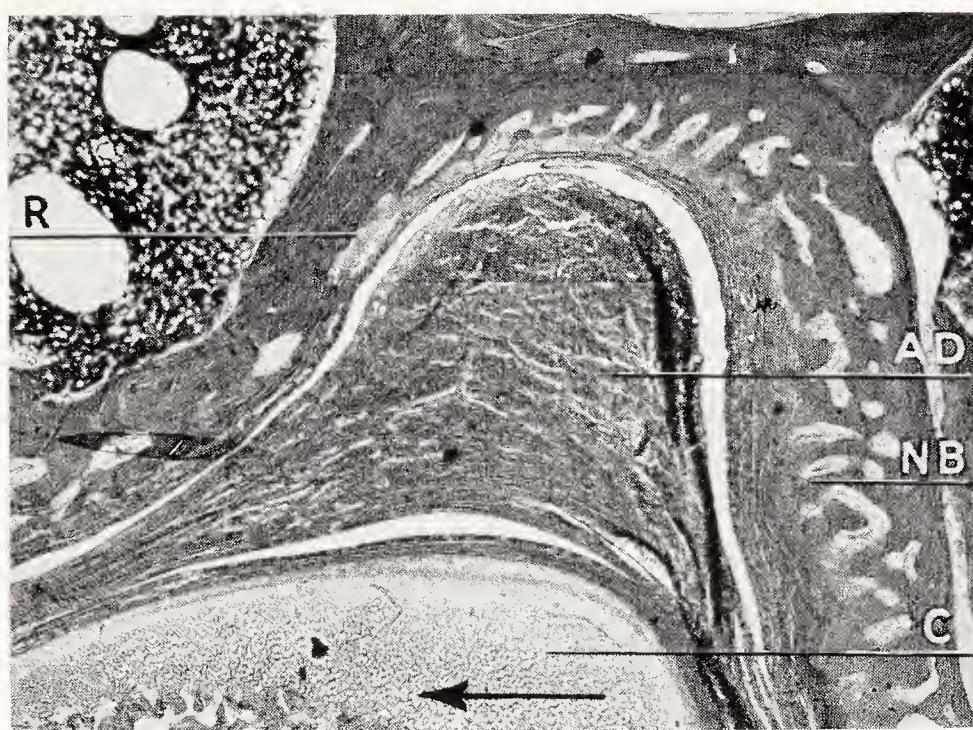


FIG. 352.—Orthodontic mesial movement of the mandible. Changes in the temporo-mandibular articulation. Monkey. *C*, condyle of mandible; the arrow indicates the direction of movement of the mandible; *AD*, articular disk; *NB*, formation of new bone on the posterior wall of the mandibular fossa; the spicules are arranged in the direction of pull; *R*, bone resorption on the anterior wall of the mandibular fossa. (Breitner, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

All teeth that had worn caps showed crushing and necrosis of the periodontal membrane, caused by the heavy occlusal force exerted upon them. Extensive root resorption was also found. These histological findings indicate that "jumping the bite" had in this case caused undue stress upon the supporting teeth and produced traumatic tissue injury.

The experiments of Breitner demonstrated that intermaxillary forces are able to produce changes in the mandibular fossa and in the architecture of the mandible. To what extent these findings are applicable to man is unknown; still, clinical observations in mesial or distal movement of the mandible indicate that the entire mandible

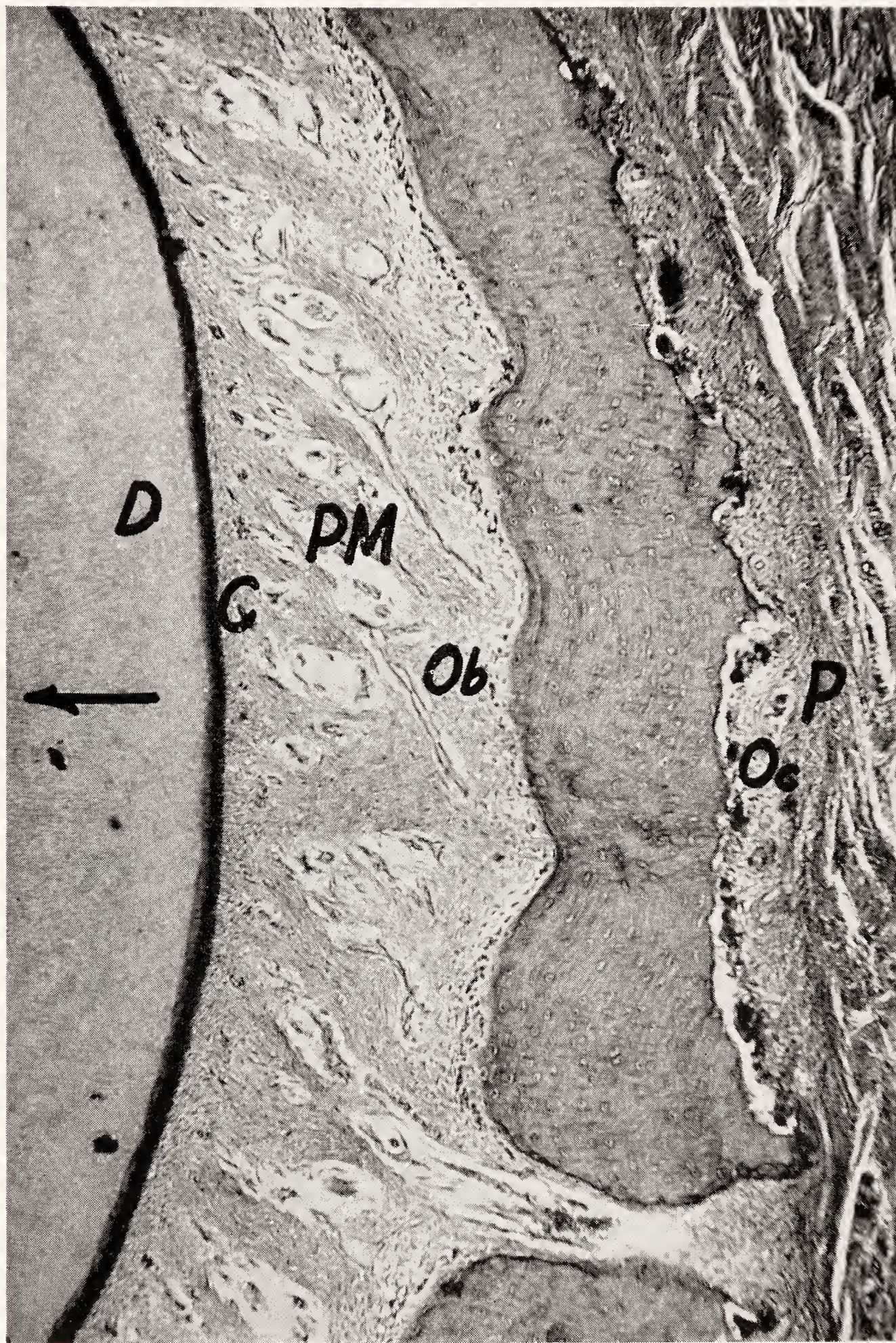


FIG. 353.—Lingual bone crest of an upper right lateral incisor that drifted labially (age, eleven years). The direction of the movement is indicated by an arrow; *P*, periosteum; *Oc*, osteoclasts on the external surface of the bone; *Ob*, osteoblasts and osteoid on the internal surface of the bone; *PM*, periodontal membrane; *C*, cementum; *D*, dentin. (Kronfeld, Angle Orth.)

may change its topographic relation to the temporo-mandibular articulation, and that rearrangement and reconstruction of the bone structure of the mandible may eventually take place as a result of orthodontic forces.

HISTOLOGICAL ANALYSIS OF THE JAWS OF A CHILD WITH MALOCCLUSION.

The author reported the histological findings in the jaws of a child, aged eleven years, whose upper right anterior teeth were crowded in such a way that the lateral incisor was displaced toward the lingual side of the central incisor, while the cuspid was erupting on the labial side of the arch. The upper left anterior teeth were in normal alignment. No treatment of any kind had been undertaken.

A comparison of sections from the left and right sides revealed that while on the normal left side the teeth were in a state of rest, on the crowded right side they were moving in an attempt to assume more normal positions. The right lateral incisor was drifting labially, and the cuspid distally. The bone changes around these teeth were essentially the same as those found by Oppenheim and Stuteville in orthodontically moved teeth. Figure 353 shows the bone and periodontal membrane on the lingual side of the drifting lateral incisor. The arrow indicates the direction in which the root was moving. New bone was being laid down on the side of the bone that faces the tooth, while osteoclasts removed the excess bone on the outer bone surface. The same tissue changes could reasonably be expected if the incisor were being moved labially by a gentle orthodontic force, instead of drifting by itself. This parallelism again emphasizes the similarity between spontaneous tooth movement, as in eruption and drifting, and artificial, orthodontic movement. The main difference between the two is that in orthodontia a more rapid movement is desirable, and therefore the forces must of necessity be greater than those acting in eruption or in drifting.

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CHAPTER XVII.

EMBEDDED TEETH. DENTIGEROUS (FOLLICULAR) CYSTS. MEDIAN ANTERIOR MAXILLARY CYSTS.

EMBEDDED TEETH.

EMBEDDED teeth are teeth that have failed to erupt and have remained partly or completely buried in the jaw tissues. Embedded teeth are said to be impacted when they are locked against an adjacent tooth in such a way that eruption is impossible.

CLINICAL CONSIDERATIONS CONCERNING EMBEDDED TEETH.

For the purpose of clinical diagnosis, it is necessary to distinguish between embedded teeth that are completely submerged and separated from the surface of the jaw by an unbroken layer of tissue, and embedded teeth the crowns of which are in communication with the oral cavity. Such partially erupted teeth are usually visible in the oral cavity; in a partially erupted lower third molar, for instance, one or two mesial cusps are often exposed, whereas the distal cusps are covered with jaw tissue. Frequently such teeth are the cause of inflammation of the surrounding soft tissues, since they permit bacteria to live and multiply in the tissues alongside the crown.

Completely embedded teeth are a continuation of the condition that is normally found in the jaws of children. The teeth are formed in the jaw as usual, but instead of erupting and taking their places in the arch, they remain within their bony crypts. The tissues overlying such completely embedded teeth may be as normal and healthy as are the tissues overlying the unerupted teeth in a child's jaw.

The incidence of embedded teeth in the upper and lower jaws has been studied by Blum. He found that in the maxilla the cuspid is most commonly embedded; next in frequency is the upper third molar, then supernumerary teeth, and very rarely incisors and second bicuspid. In the mandible it is generally the third molar that is embedded; much more rarely are the lower cuspids and bicuspid embedded. From Blum's material on both maxillary and mandibular embedded teeth, the following frequency percentages can be calculated: third molars, 64 per cent; cuspids, 24 per cent; second

bicuspid, 4 per cent; supernumerary teeth, 3 per cent; all other teeth combined, 5 per cent.

Sometimes the same tooth on the right and left side is embedded in the same patient, for instance, both lower third molars or both upper cuspids.

Etiology of Embedded Teeth.—The factors that may cause teeth to remain embedded can be classified as follows:

A. Primary Displacement of Tooth Germ:

Excessive distance from point of eruption.

Abnormal position.

B. Disturbances of Eruption:

Malformation of tooth: Abnormal size or shape of crown or root.

Lack of space.

Disturbances during formative period.

Supernumerary teeth or tumors.

C. Fusion Between Tooth and Jaw Bone.

D. Systemic Causes:

Heredity.

Endocrine disturbances.

Cleidocranial dysostosis.

A. Primary Displacement of Tooth Germ.—*Excessive Distance from Point of Eruption.*—A tooth germ may, for unknown reasons, develop in an area remote from its normal location. Lower third molars, for instance, may be located in the ramus of the mandible or even near the coronoid or condyloid process; upper cuspids or third molars may be situated in the lateral wall of the maxillary sinus. Such teeth usually remain embedded, since they cannot reach their normal place of eruption (Fig. 354).

Abnormal Position of Tooth Germ.—Occasionally a tooth germ develops at an extreme mesial, distal, labial, or lingual inclination, so that the tooth lies horizontally in the jaw. Even complete inversion occurs: the root points occlusally and the crown is directed away from the jaw surface. After such a tooth is formed, it lies in the jaw in a position that makes eruption into the oral cavity impossible. Sometimes inverted teeth erupt into the nose or into the maxillary sinus, or they remain embedded in their abnormal position. The cause for such malpositions of tooth germs is unknown.

B. Disturbances of Eruption.—*Malformation of the Tooth.*—A gross abnormality or deformity of crown or root may prevent a tooth from erupting. Such deformities are excessively large crowns, malformed crowns, and fused teeth. Abnormal shape of the root, especially a

crooked root that becomes locked against the roots of adjacent teeth, can also interfere with eruption.

Lack of Space.—Lack of sufficient space in the dental arch is one of the main causes of embedded teeth. This can be caused by a discrepancy between jaw size and tooth size. If an individual inherits a small jaw from one parent and large teeth from the other, there may not be enough space in the jaw to permit the dental arch to form normally. The last teeth to erupt—cuspids and third molars—are either forced out of line, or they fail to erupt.



FIG. 354.—Radiograph showing misplaced mandibular third molar. (Cryer.)

In modern man there seems to be a general tendency toward underdevelopment and shortening of the lower jaw. This accounts for many of the impactions of lower third molars.

Premature loss of deciduous teeth is another cause of lack of space and subsequent tooth impaction. The teeth most frequently involved are the second bicuspid. If the second deciduous molar is lost several years before the bicuspid is due to erupt, the first bicuspid drifts or tilts distally and the first permanent molar mesially, so that the second bicuspid cannot erupt and remains embedded (Fig. 355). If later the first bicuspid and the first molar are moved apart orthodontically, the embedded bicuspid usually erupts normally.

Disturbances During the Formative Period.—Severe jaw infections or osteomyelitis in childhood produce abnormal form of the jaws, bone scars, and resorptive processes in the developing tooth germ. These may later prevent eruption. The same is true of fractures of the jaws or other injuries involving the tooth germs. Teeth located

in or near a line of fracture in the jaw of a young child may fail to erupt either because the tooth germ itself was damaged or because the bony crypt was injured and the normal relation of the tooth to the surrounding tissues disturbed.

In a cystic detachment of the enamel epithelium from the crown and formation of a dentigerous cyst, eruption is impossible as long as the cyst is present. After an operation in which the cyst is opened and the pressure of the cyst fluid relieved, the tooth may follow its natural tendency to move in an occlusal direction and may erupt (see Figs. 380–383).

Supernumerary Teeth and Tumors.—The presence of supernumerary teeth is often a cause for the failure of teeth to erupt. There

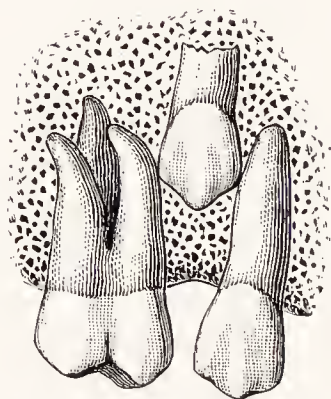


FIG. 355.—The effects of the premature loss of a deciduous second molar. (Burchard and Inglis.)

have been cases in which a supernumerary tooth between the upper central incisors kept both incisors from erupting, as there was not enough space between the lateral incisors for the eruption of three teeth. The surgical removal of the supernumerary tooth is often followed by the normal eruption of the other embedded teeth.

In a similar manner a dental tumor (odontoma) may lie close to a normal tooth and prevent its eruption. Removal of the obstacle usually brings about eruption.

C. Fusion Between Tooth and Jaw Bone.—A bony union between a tooth germ and the jaw bone may result from traumatic injuries to the tooth germ or from inflammation of the bone. The usual sequence of events is that the unerupted tooth is partly resorbed and then, during the reparative phase, becomes united with the bone. Sometimes the reason for resorption of a tooth germ is not known.

D. Systemic Causes.—*Heredity.*—Heredity plays an important part in the occurrence of embedded teeth. Every practitioner knows of instances in which the same type of embedded tooth has occurred in members of two, even three, generations. It is not definitely known exactly what is inherited, whether it is the small size of the jaw or the large size of the tooth, or a certain type of malalignment of the tooth germs. The fact remains, however, that embedded teeth are often a family characteristic.

Endocrine Dysfunction.—Several of the endocrine glands have an influence upon the eruption of the teeth. However, disturbances of eruption may not be noticeable clinically except in extreme cases

of endocrine dysfunction. In cretins, for instance, the eruption of the teeth is greatly retarded and many teeth remain embedded.

Cleidocranial Dysostosis.—Cleidocranial or cleidofacial dysostosis is characterized by congenital defects in the skeleton. The bones most commonly affected are the skull and the clavicles. The skull is slightly enlarged; the ossification of the skull bones and the closure of the sutures are greatly delayed, so that open fontanelles are sometimes found in adults. The paranasal sinuses are abnormally small or missing. The mental development, as a rule, is not disturbed. Either the clavicles are absent, or short stumps of them may be present at the acromial or sternal end. Such a person is able to bring the shoulders almost or completely in contact in front of the chest, since there are no clavicles to hold the shoulders from the chest wall.

Invariably individuals with cleidocranial dysostosis have many embedded teeth. The deciduous teeth usually erupt fairly normally, but they are not shed at the proper time. Sometimes a few permanent posterior teeth erupt, but the rest remain embedded; however, there are cases on record in which not a single permanent tooth had erupted. Radiographic examination shows that the teeth are formed and in normal position, but evidently the stimulus for eruption is absent.

Cleidocranial dysostosis is a dominant, hereditary characteristic without sex linkage. A typical example is the family recently observed by Heupel. In this family, consisting of father, mother, and eight children (3 boys and 5 girls), the father and the three youngest girls had cleidocranial dysostosis. The father had been wearing full dentures since childhood, although a radiographic examination of his jaws showed that almost all of the permanent teeth were present, embedded in the jaw bone. In all three girls the clavicles were either absent or only short rudiments; a few permanent teeth had erupted, but most of them, as well as several supernumerary teeth, were embedded.

Embedded Upper Cuspids.—In the upper jaw the cuspids are the teeth that are most frequently embedded. An embedded cuspid can occupy any position from the vertical to the horizontal. In a vertical impaction the usual cause is lack of space between lateral incisor and first bicuspid; the crown of the cuspid is wedged in between the roots of these two teeth, making eruption impossible. In an angular impaction the tip of the cuspid crown impinges upon the root of the lateral or central incisor, sometimes causing root resorption of the latter. In a horizontal impaction the cuspid may

be located high in the jaw; its crown lies near the central or lateral incisor, its apex near the root ends of the second bicuspid or first molar (Fig. 356).

Sometimes an embedded cuspid is located entirely on the lingual side of the dental arch, buried in the bone of the hard palate; again, it may be situated near the labial bone surface, so that its crown can sometimes be felt beneath the intact mucous membrane of the vestibule. On rare occasions embedded cuspids have been found in the walls of the nasal cavity or the maxillary sinus.

If a permanent cuspid is embedded, the deciduous cuspid may remain in place until the individual is thirty or forty years old. Then it usually becomes loose and falls out.

As a rule, embedded cuspids cause no clinical symptoms whatever. The majority of them are discovered accidentally by radiographs. Aside from the effect upon the arch and the malocclusion that may be caused by the absence of the cuspid, the only serious consequence is the possibility of root resorption of the incisors, especially of the lateral incisor.

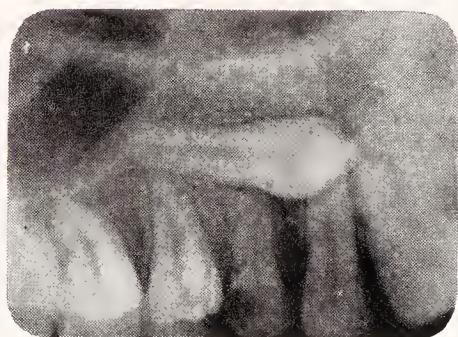


FIG. 356

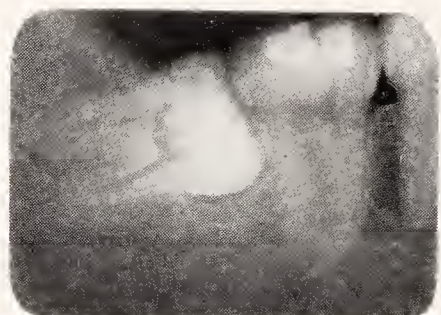


FIG. 357

FIG. 356.—Radiograph of an embedded upper cuspid. The tooth is lying horizontally in the maxilla; the tip of the crown is near the root of the central incisor, the apex near the roots of the bicuspids. The deciduous cuspid is still in place. (Ennis.)

FIG. 357.—Radiograph of an impacted lower third molar. The third molar lies in a horizontal position, its crown impinging upon the distal surface of the second molar, its roots extending into the ramus. (Ennis.)

Embedded Lower Third Molars.—In the lower jaw the third molars are the teeth most frequently embedded. The primary reason for the frequency of the third molar impaction is lack of space: often there is not enough room between the distal surface of the second molar and the anterior margin of the ascending ramus, and as a result, the third molar cannot erupt. Winter has given the following classification of third molar impaction:

Vertical Impaction.—In vertical impaction the third molar is in a normal upright position; the entire crown or the distal portion of the crown is covered by soft tissue and bone, or sometimes by soft

tissue only. This type of impaction is undoubtedly the result of lack of space in the mandible.

Horizontal Impaction.—In a horizontal impaction the third molar lies horizontally in the arch. Its occlusal surface is usually in contact with the distal surface of the second molar, its roots pointing distally in the ramus (Fig. 357).



FIG. 358.—A common form of impacted lower third molars. (Cryer.)

Distoangular Impaction.—In a distoangular impaction, a rather uncommon form of impaction, the tooth is in an upright position, with the crown pointing distally toward the ramus, and the roots mesially toward the roots of the second molar.

Mesioangular Impaction.—A mesioangular impaction is the most common type of impaction of the third molar (Figs. 358, 359 and 360). The tooth points forward at an angle of from 30 to 60 degrees; its mesial cusps are in contact with the second molar; its distal cusps are free. The area where the second and third molar are in contact depends largely upon the angulation of the latter: the more the impaction approaches the vertical type, the higher up against the crown of the second molar will the third molar rest.

In addition to the angulation of the third molar in the sagittal plane, there is also a possibility of a deflection in the frontal plane. Accordingly, Winter distinguishes between buccal and lingual deflection. In buccal deflection the crown of the impacted third molar deviates toward the buccal side of second molar; in lingual deflection



FIG. 359.—Internal bone plate removed. (Cryer.)

toward the lingual side. In extreme cases of deflection, a lower third molar may lie in a transverse position in the mandible, its crown pointing toward the buccal or the lingual side. Then the usual intraoral radiograph shows only an occlusal view of the crown, since the direction of the rays coincides with the axis of the molar (Fig. 361).

Lower third molars may be completely embedded or partially erupted. In a horizontal or in distoangular impaction, the tooth is



FIG. 360.—External bone plate removed. (Cryer.)

likely to be completely buried in the jaw; in a vertical or mesioangular impaction, one or both of the mesial cusps are usually erupted or at least accessible from the oral cavity. In the latter case the tissues surrounding the embedded tooth are frequently infected or inflamed; occasionally grave infections and osteomyelitis of

the jaw arise from the investing tissues of partially embedded lower third molars.

Embedded Bicuspids.—Bicuspids are sometimes found fully embedded. They usually lie in a horizontal position in the jaw, with the crown pointing mesially or distally (Fig. 362).

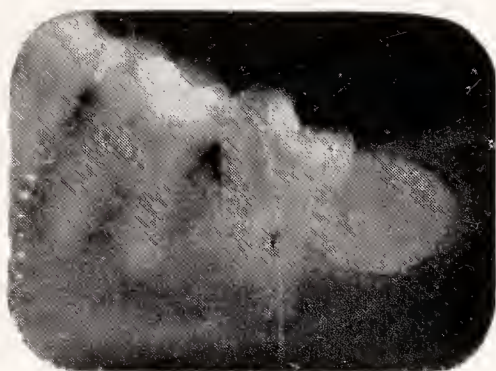


FIG. 361

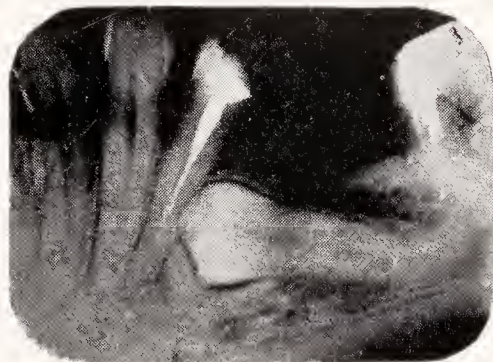


FIG. 362

FIG. 361.—Transverse, horizontal impaction of lower third molar. (Ennis.)

FIG. 362.—Radiograph of an impacted lower bicuspid. (Ennis.)



FIG. 363



FIG. 364

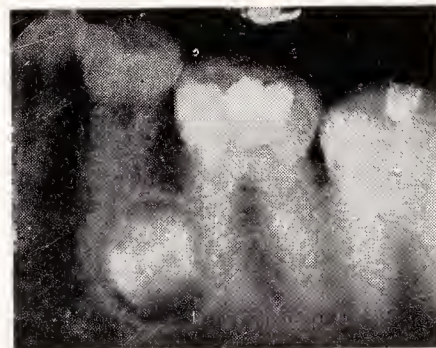


FIG. 365

FIGS. 363 to 365.—Radiographs of embedded supernumerary teeth.

FIG. 363.—Peg-shaped supernumerary tooth between the upper central incisors. The axis of the supernumerary tooth lies in a labio-lingual direction. The surrounding tissues are intact.

FIG. 364.—Supernumerary tooth in the cuspid region interfering with the eruption of the permanent cuspid.

FIG. 365.—Supernumerary tooth (third bicuspid) in the region of the lower second bicuspid. The bicuspids are erupted and in normal occlusion. Of the supernumerary tooth, only the crown is formed.

Embedded Supernumerary Teeth.—Supernumerary teeth are frequently embedded. In the region of the upper central incisors the radiograph sometimes reveals small peg-shaped, impacted, supernumerary teeth; these may cause a diastema between the upper central incisors (Figs. 363 and 364). Embedded supernumerary bicuspids are occasionally found in the lower jaw (Fig. 365), and on rare occasions an embedded fourth molar is found on the distal side of the normal third molar (see Fig. 13).

Treatment of Embedded Teeth.—There are three ways to deal with embedded teeth: their eruption may be made possible; they

may be removed surgically; or they may be left alone. The first possibility applies mostly to cuspids and bicuspids. With embedded upper cuspids it is sometimes possible to expose the crown surgically, to drill a small hole into the enamel, and to cement a hook into this hole. To this hook is applied an orthodontic force, usually by means of rubber bands, and thus the embedded tooth is gradually moved in the direction of the occlusal plane (Fig. 366). Of course, it is necessary first to create sufficient space in the arch, so that the crown of the cuspid can occupy its proper position. If a bicuspid



FIG. 366.—Radiographs of embedded cuspids that are being brought to place by means of orthodontic appliances. (McCoy.)

is kept from erupting because of premature loss of the deciduous predecessor and subsequent closing of the space, spontaneous eruption will often take place after the lost space is restored by orthodontic treatment (Erickson).

Surgical removal of an embedded tooth is indicated if the tooth causes pain or discomfort, root resorption or malalignment of adjacent teeth, or, in case of partially embedded teeth, inflammation of the surrounding tissues. A discussion of the various operations used for this purpose is not within the scope of this book.

Finally, it may be indicated to leave undisturbed a fully embedded, symptomless tooth. In all probability such a tooth is harmless, and the difficulties and possible complications resulting from its removal may far outweigh any advantages gained by it. The work of Logan has clearly illustrated that a fully embedded tooth cannot *per se* be considered a source of infection. A similar viewpoint has been expressed by Austin: "We believe that unerupted teeth that are lying within the body of the bony tissue, and apart from the neighboring teeth, and that do not cause any local symptoms, can be retained with little danger to the host."

CLINICAL SYMPTOMS CAUSED BY EMBEDDED TEETH.

The subjective symptoms that may be caused by embedded teeth can be divided into those due to pressure and those due to inflammation. Embedded teeth have an inherent tendency to move in an occlusal direction. If they meet an obstacle, such as an embedded lower third molar impinging upon the root of the second molar, they exert pressure upon this obstacle and cause pain. An embedded tooth may also impinge upon a nerve and cause pain of a neuralgic type.

Another result of the pressure exerted by an embedded tooth may be changes in the alignment of the other teeth. Embedded lower third molars have been known to cause mesial displacement of molars and bicuspid and crowding of the anterior teeth. An embedded upper cuspid may press against the root of an adjacent lateral incisor and cause the latter to change its position.

Partially erupted teeth are frequently the cause of inflammation of the surrounding tissues. Especially around lower third molars severe inflammation is sometimes observed, with extensive cellulitis and abscess formation. The usual history is one of repeated, intermittent attacks of pain and swelling, with some discharge of pus from the soft tissues alongside the tooth. The soft tissue flap overlying the crown becomes swollen and sore. Fever, trismus (difficulty in opening the mouth), and lymphadenitis may accompany these attacks. The radiograph often reveals bone destruction, especially on the distal side of the third molar crown.

Third Dentition.—From time to time newspapers report that an elderly person who has been edentulous for many years is cutting a "third" set of teeth. Such reports are based upon the fact that embedded teeth, belonging to the permanent dentition, are sometimes brought to the surface either by a belated process of eruption or by the advancing senile atrophy of the jaw bone. The pressure caused by a denture may be one of the factors that accelerate bone

resorption and thus tend to expose a formerly deeply buried tooth. The existence of a third dentition could be proved only if all teeth of the second dentition were erupted and radiographs showed that tooth germs were again forming within the jaws. To the author's knowledge, such a true third dentition has never been reported by anyone qualified to make an accurate examination and correct diagnosis.

MICROSCOPIC EXAMINATION OF EMBEDDED TEETH WITH THEIR SURROUNDING TISSUES.

In order to obtain positive information about the relationship between embedded teeth and their surrounding structures, human

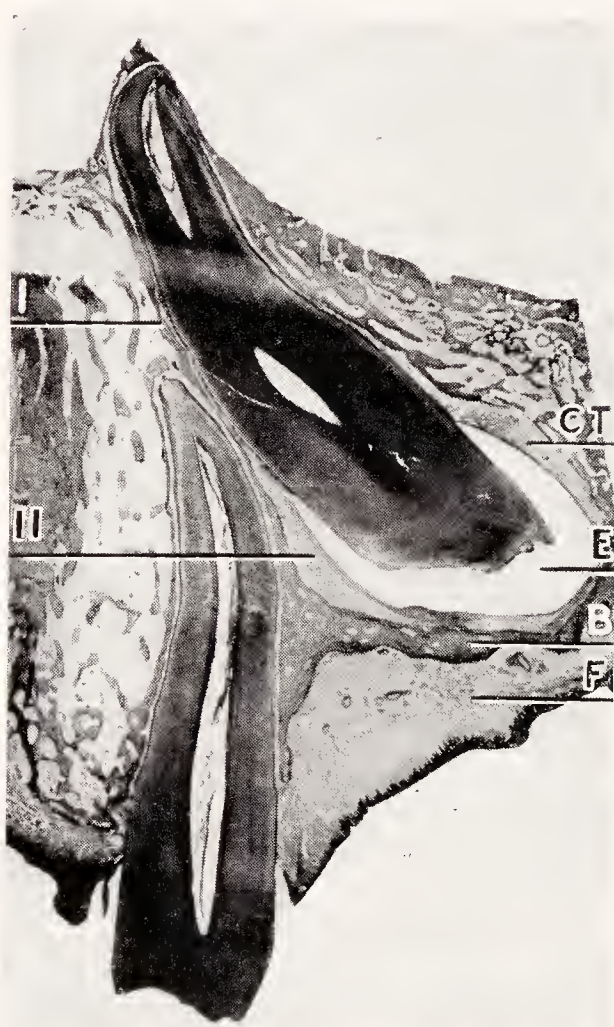


FIG. 367.—Mesio-distal section through an embedded upper cuspid and an erupted first bicuspid. *E*, enamel of embedded cuspid; *CT*, connective tissue capsule surrounding the crown of the cuspid; *B*, bone covering the cuspid; *F*, fibrous tissue of the edentulous ridge overlying the cuspid. Higher magnifications of areas *I* and *II* are shown in Figures 368 and 369. (Gottlieb, *Fortschr. d. Zhk.*, courtesy of Georg Thieme, Leipzig.)

jaws that contain such teeth must be sectioned and examined microscopically. A few specimens of this kind will be described here. Figure 367 shows a mesio-distal section through the right maxilla of a young adult. The first bicuspid has erupted normally, but the right upper cuspid is completely embedded and lies in the maxilla at an angle of about 45 degrees, with the crown pointing downward against the lateral incisor and the apex in a bony ridge on the floor of the maxillary sinus. The crown of the cuspid is surrounded by a layer of fibrous connective tissue. The alveolar ridge over the impacted cuspid is covered with bone and normal mucosa; the deciduous cuspid has been lost. Special attention should be called to the wide space filled with connective tissue between enamel and alveolar bone, a space which appears in the radiograph as a dark area surrounding the crown of the impacted tooth. The distance between root surface and bone,

however, is very small. In Figures 368 and 369, higher magnifications of different areas from the surface of this impacted cuspid are

reproduced. Figure 368 corresponds to area *I* in Figure 367. The periodontal membrane is thin and atrophic, characteristic of non-functioning teeth (see page 362). Its thickness is about 0.06 to 0.08 mm. Figure 369 illustrates area *II* of Figure 367, on the surface of the crown. The enamel is in organic connection with the enamel epithelium, a condition always found in teeth before eruption (see page 289). The distance between enamel surface and alveolar bone is about ten times the distance between cementum and bone in Figure 368. Dense, fibrous connective tissue is arranged parallel to the surface of the crown.

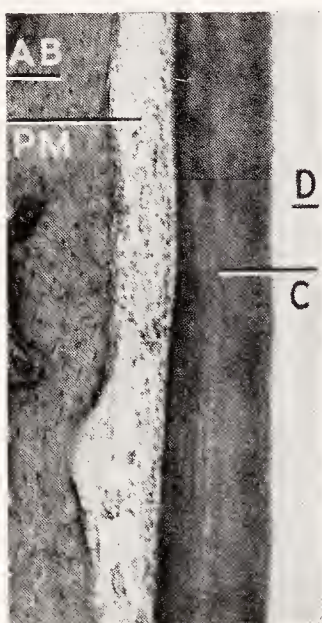


FIG. 368

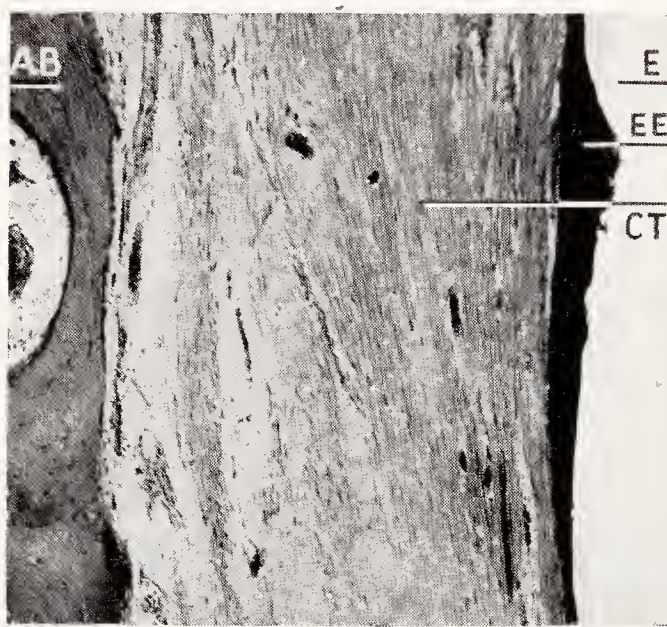


FIG. 369

FIG. 368.—High magnification of area *I* in Figure 367. *AB*, alveolar bone; *PM*, periodontal membrane; *C*, cementum; *D*, dentin. The periodontal membrane consists of loose connective tissue (lack of functional stimulus). Average thickness of periodontal membrane, 0.06 mm.

FIG. 369.—High magnification of area *II* in Figure 367. *AB*, alveolar bone; *CT*, fibrous connective tissue encapsulating the crown; *EE*, enamel epithelium; *E*, enamel. Distance between enamel and alveolar bone, 0.7 mm.

In radiographs the dark space around the crowns of embedded teeth has frequently been misinterpreted. It has incorrectly been spoken of as an area of infection or as a cystic formation, because of lack of suitable material on which to study the tissues surrounding embedded teeth. Now it is known that this dark area around the crown is merely the shadow of the connective tissue of the tooth follicle; it corresponds to the dark area surrounding every tooth germ prior to eruption.

A completely embedded lower bicuspid is illustrated in Figure 370. The tooth lies almost horizontally in the body of the mandible, the crown being located slightly higher than the root but still separated from the surface of the jaw by a bony plate. The enamel is surrounded by a layer of connective tissue underneath which the man-

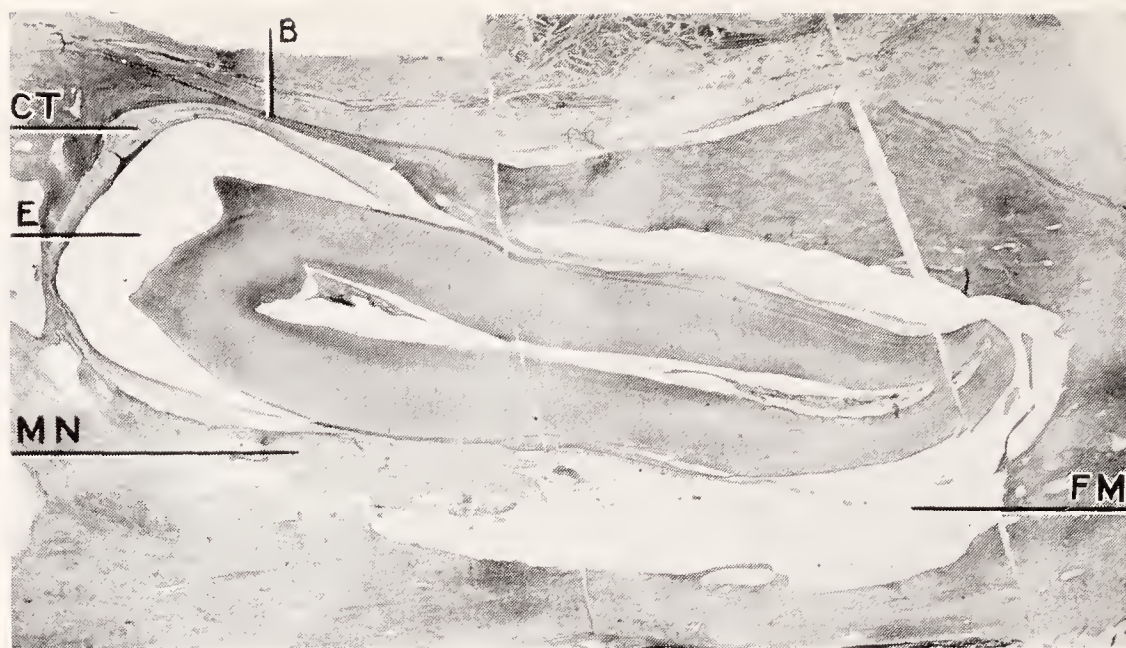


FIG. 370.—Embedded lower bicuspid lying horizontally in an edentulous mandible. *E*, enamel of bicuspid; *CT*, connective tissue surrounding the crown; *B*, bone covering the crown; *MN*, mandibular nerve located below the tooth; *FM*, fat marrow. (Kellner, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

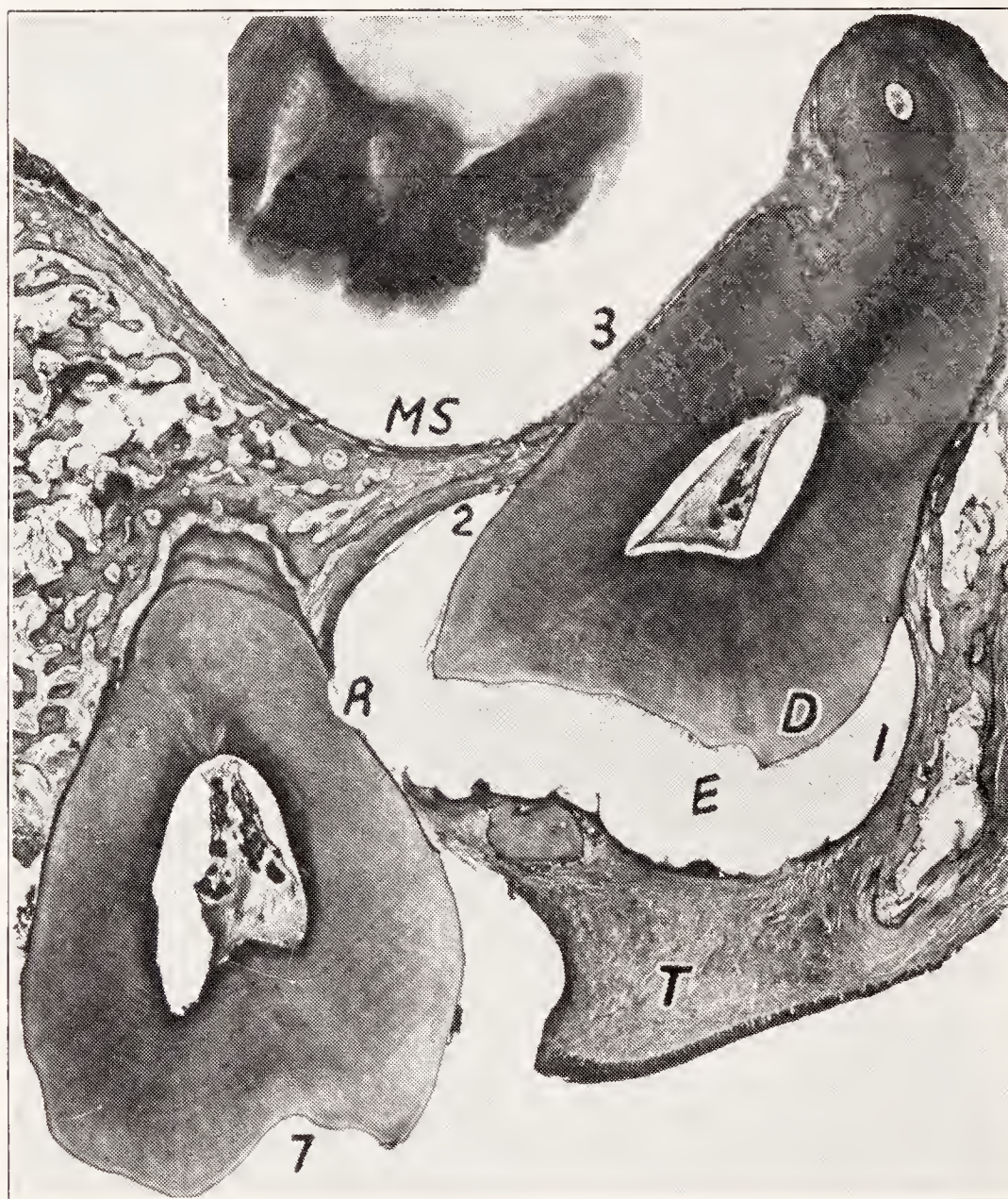


FIG. 371.—Mesio-distal section through an erupted upper right second molar and completely embedded third molar; man, thirty-two years old. The radiograph taken before decalcification of the specimen shows the upper molars and the large antrum. *T*, connective tissue of maxillary tuberosity; *E*, enamel space; *D*, dentin of the fully embedded third molar; *MS*, maxillary sinus; *7*, crown of second molar; *R*, small area of resorption on distal surface of root of second molar. (Logan, *Jour. Am. Dent. Assn.*)

dibular nerve can be seen. The root is surrounded by a thin lamella of bone. In some areas the root surface of the tooth is in direct contact with the fat marrow of the mandible. The root is covered with a thick layer of cementum.

The histological findings in several completely embedded molars have been reported by Logan. His investigation was primarily concerned with the question of whether or not the pulps and investing tissues of such teeth are infected. Starting with the premise that infection produces a characteristic tissue reaction, namely, inflam-

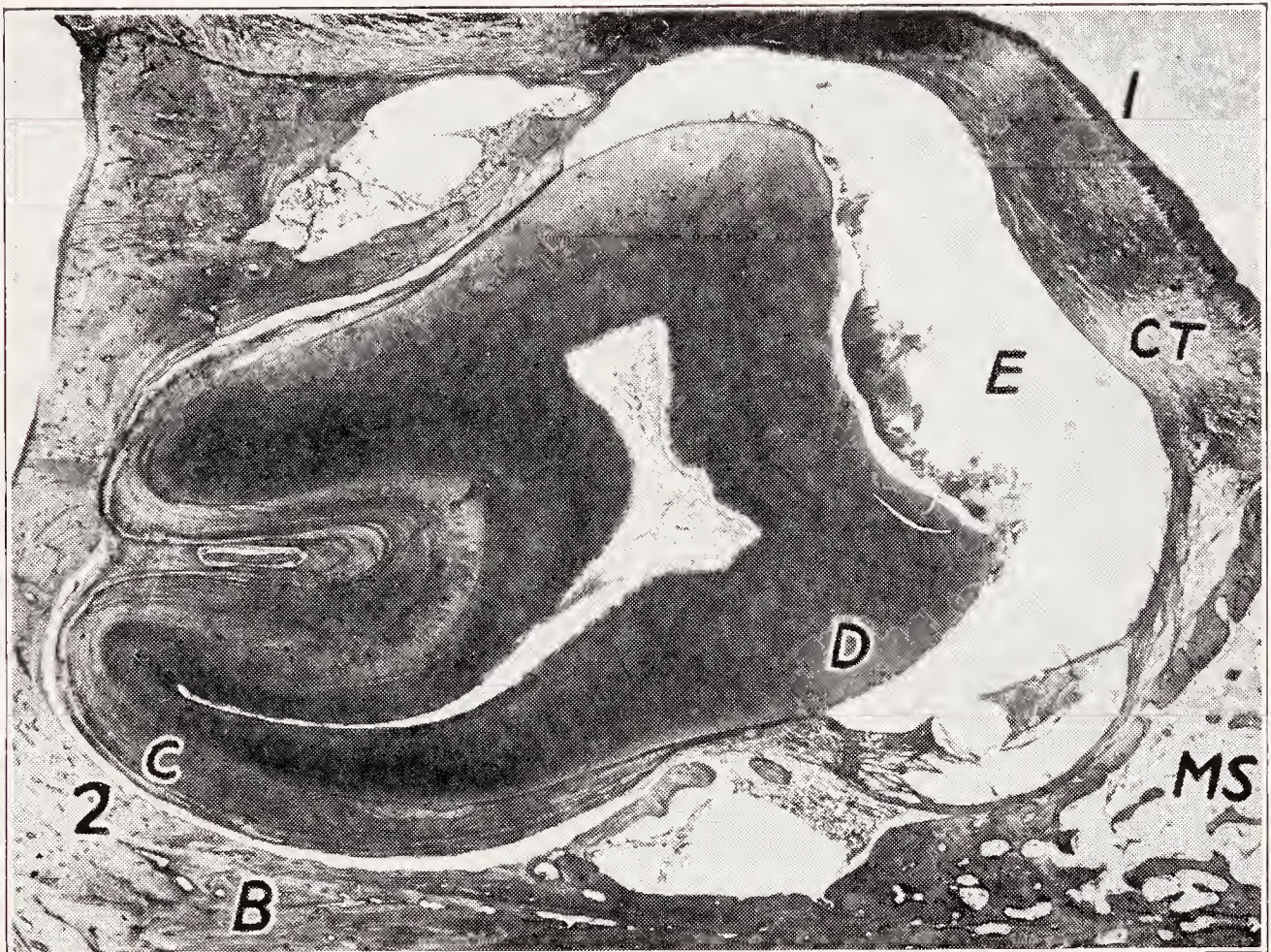


FIG. 372.—Mesio-distal section through the completely embedded lower left third molar of a man, thirty-eight years old. *CT*, oral mucosa and connective tissue covering crown; *E*, enamel space; *D*, dentin; *C*, thick layers of cementum; *B*, bone; *MS*, marrow spaces. The area marked 1 is shown in Figure 373 in higher magnification. (Logan, Jour. Am. Dent. Assn.)

mation, he studied microscopically the pulps and tooth follicles of embedded teeth to ascertain the presence or absence of such a reaction. Figure 371 shows a section through a completely embedded, impacted upper third molar and the adjacent erupted second molar. The individual from whom this specimen was obtained had a very large maxillary sinus, as can be seen in the radiograph; the posterior wall of the sinus forms the mesial wall of the socket of the embedded tooth. At the point where the crown of the third molar impinges upon the root of the second molar, the latter shows a shallow area

of resorption. The connective tissue surrounding the embedded tooth is free of inflammatory changes. The pulp is atrophic and contains numerous pulp stones, a condition also found in all of the other teeth of this individual.

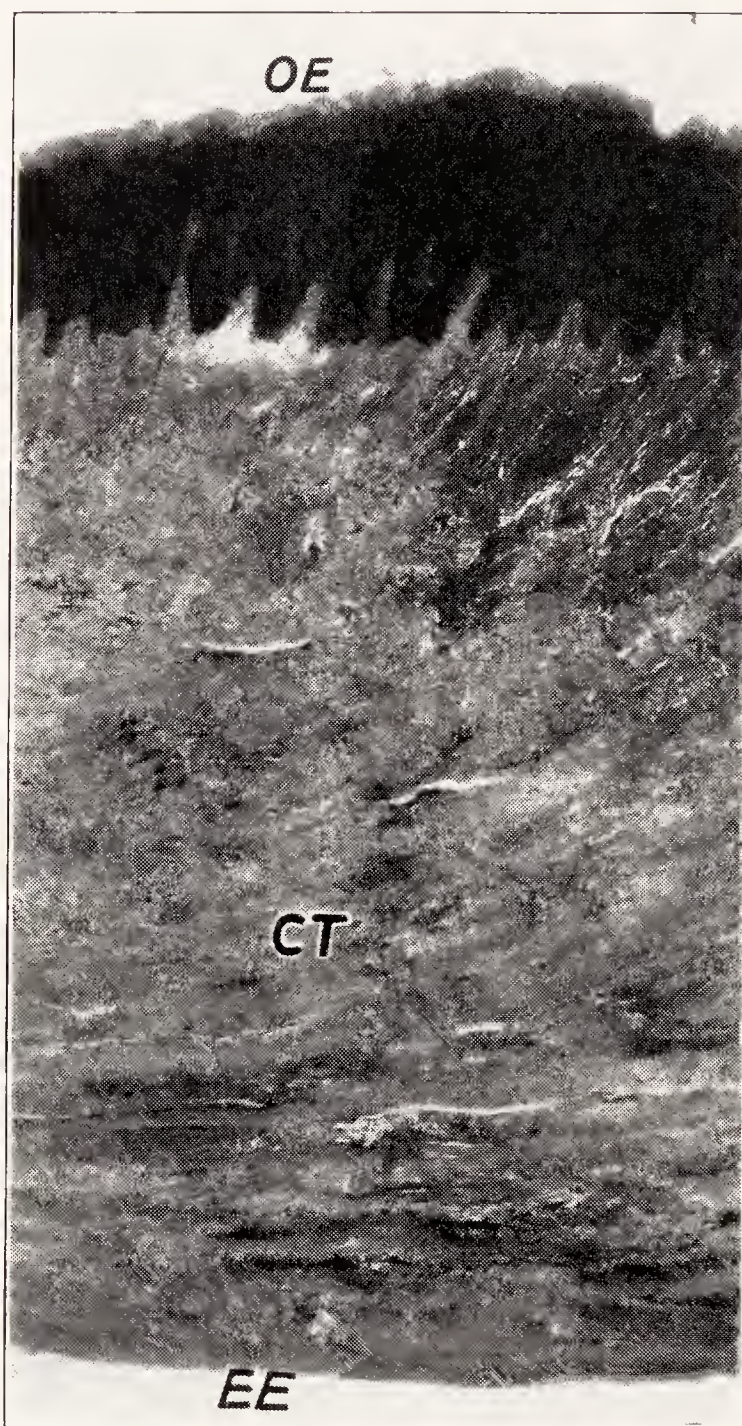


FIG. 373.—Higher magnification of area 1 in Figure 372. Tissue overlying crown of completely embedded tooth. *OE*, oral epithelium; *CT*, dense fibrous connective tissue of lamina propria and tooth follicle; *EE*, enamel epithelium. (Logan, Jour. Am. Dent. Assn.)

A completely embedded lower third molar, located in the edentulous posterior portion of the mandible of a man, aged thirty-eight years, was also reported by Logan (Fig. 372). The periodontal membrane is thin and atrophic, its average thickness being 0.08 mm. Toward the jaw surface the crown is covered with fibrous connective

tissue and normal mucosa; no evidence of inflammation was found in this tissue (Fig. 373).

From the findings in these and other completely embedded teeth, Logan came to the conclusion that such teeth merely represent a continuation of the pre-eruptive state. Since there is no evidence that a fully embedded tooth surrounded by clinically and radiographically normal tissues is infected, the profession should be warned against expecting benefit from the removal of such teeth in patients suffering from systemic diseases, possibly originating in or caused by focal infection. However, the presence of pain, pressure against adjacent teeth, root resorption, and cyst-formation are definite indications for the removal of completely embedded teeth, even though infection is absent.



FIG. 374.—Deposition of cementum upon the enamel of an embedded lower third molar. *D*, dentin; *E*, enamel; *CEJ*, cemento-enamel junction; *Cu*, enamel cuticle; *C*, cementum on the surface of the enamel; *CT*, connective tissue.

Deposition of Cementum Upon the Crowns of Embedded Teeth.—

As long as the enamel epithelium covering the crown of an embedded tooth is intact, the enamel is separated from the connective tissue of the tooth follicle, and no changes can take place on the enamel surface. If, however, the tooth remains embedded over a long period of time, the enamel epithelium may become atrophic and disappear. Then the connective tissue of the tooth follicle may deposit cementum directly upon the enamel surface. Frequently this deposition of cementum occurs near the cemento-enamel junction; then the cementum extends in the form of tongues or spurs toward the crown (Fig. 374). Sometimes cementum forms on the occlusal surface of embedded molars.

RESORPTION OF EMBEDDED TEETH.

Resorptive processes in the crown or root of embedded teeth are not uncommon. Their cause is unknown. The resorption of enamel and dentin is almost invariably followed by a deposition of cementum upon the resorbed surfaces and by an ingrowth of cancellous bone (Fig. 375). In advanced cases of resorption of embedded teeth, most of the tooth structure is replaced by spongy bone and bone

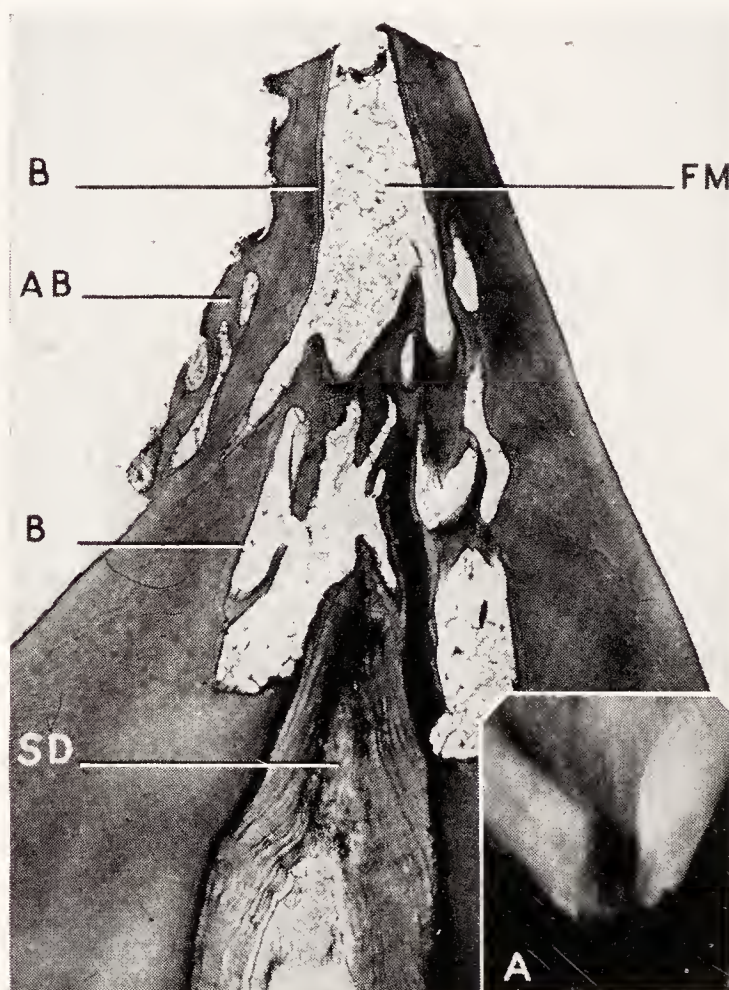


FIG. 375.—Resorption of the crown of a completely embedded upper cuspid. *A*: Radiograph of two embedded cuspids in an edentulous maxilla. Both cuspids show evidence of resorption of the crown. The histological specimen is the left cuspid. *FM*, fat marrow occupying the spaces created by dentin resorption; *B*, deposits of bone upon the resorbed dentin; *AB*, alveolar bone united with the crown; *SD*, secondary dentin in the tip of the pulp chamber.

marrow, and a histological examination reveals only scattered islands of dentin within the bone. In the radiograph a resorbed embedded tooth has a typical appearance: instead of having a clear, dense outline, the surface appears uneven, and sometimes large portions of the crown or root are missing, having been replaced by connective tissue and spongy bone. Such a tooth has a rough, eroded surface, and the defects may look like caries. A microscopic examination, however, reveals beyond doubt that the loss of tooth structure is due to resorption and not to caries (Fig. 245).

In the description of radiographs of embedded teeth, areas of resorption in the crown of an embedded tooth are sometimes spoken of as areas of decalcification. This term is vague and misleading because it implies a non-existing similarity between these areas of resorption and dental caries. *A completely embedded tooth cannot decay.* Dental caries is impossible without the presence of oral fluids and oral microorganisms, and neither have access to a completely embedded tooth. The only way in which such a tooth can change is by the action of osteoclasts (resorption).

DENTIGEROUS (FOLLICULAR) CYSTS.

A dentigerous or follicular cyst is a cyst within the jaw containing the crown of an unerupted tooth. Such cysts result from an accumulation of fluid between the enamel and the surrounding soft

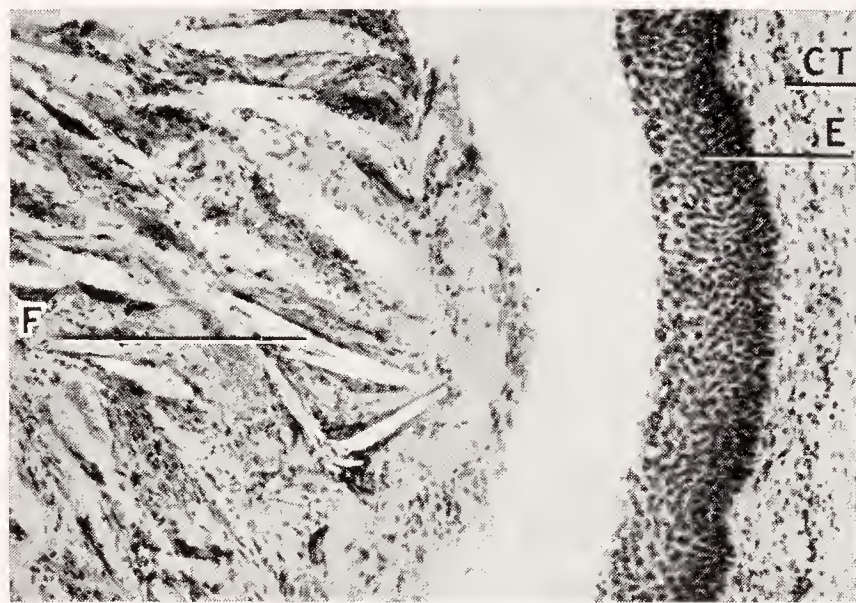


FIG. 376.—Part of the wall of a dentigerous cyst. *E*, cyst epithelium; *CT*, connective tissue of the cyst wall; *F*, coagulated fibrin, wandering cells, and cholesterol crystals in the cyst cavity.

tissues. Normally the enamel surface of an unerupted tooth is united with the enamel epithelium, but if this junction is severed, fluid is discharged into the resulting space around the crown; subsequently the surrounding tissues become distended and form a cystic sac. The soft tissue investing an unerupted tooth is called the tooth follicle; therefore, dentigerous cysts are also called follicular cysts. The crown projects into the cyst sac, which is attached to the tooth at the border between crown and root. The cyst is lined with squamous epithelium derived from the enamel epithelium; it is filled with a clear, serous, yellowish fluid frequently containing crystals of cholesterol (Fig. 376). The tooth within the cyst may present various stages of development: if the cyst develops early

in life the crown only may be present; if it develops later in life, a part of the root or the entire root may be formed.

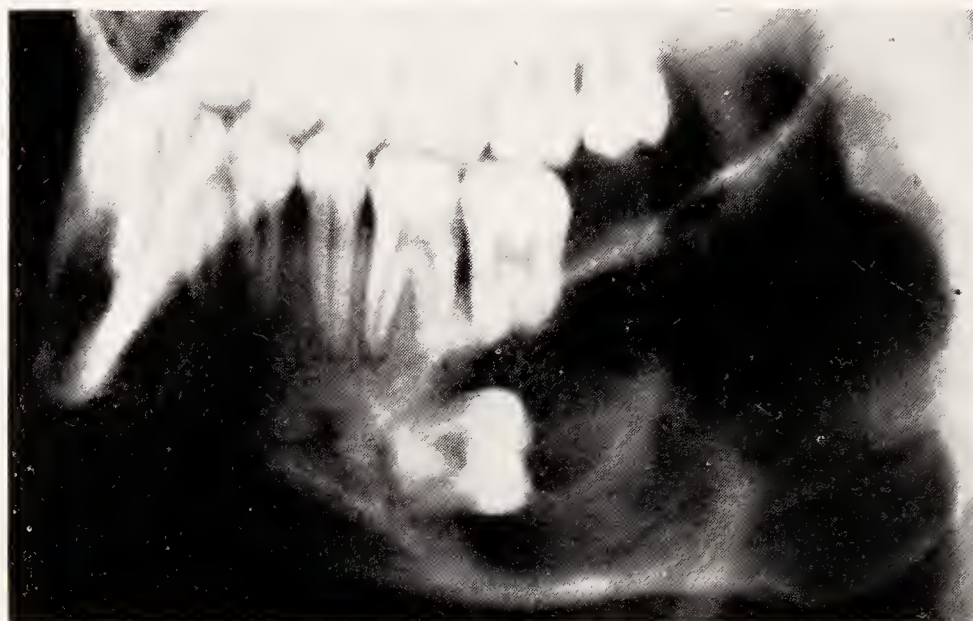


FIG. 377.—Radiograph of a large dentigerous cyst involving angle and ramus of the mandible. The cyst developed from the lower third molar which is displaced below the roots of the first and second molar.

The bone around a dentigerous cyst is resorbed by the pressure of the cyst sac; thus large areas of bone destruction may result (Figs. 377 and 378). A typical dentigerous cyst grows slowly without causing any pain or discomfort; its presence is usually discovered either by the absence of a tooth or by a swelling on the surface of the jaw. So long as the cyst is intact,

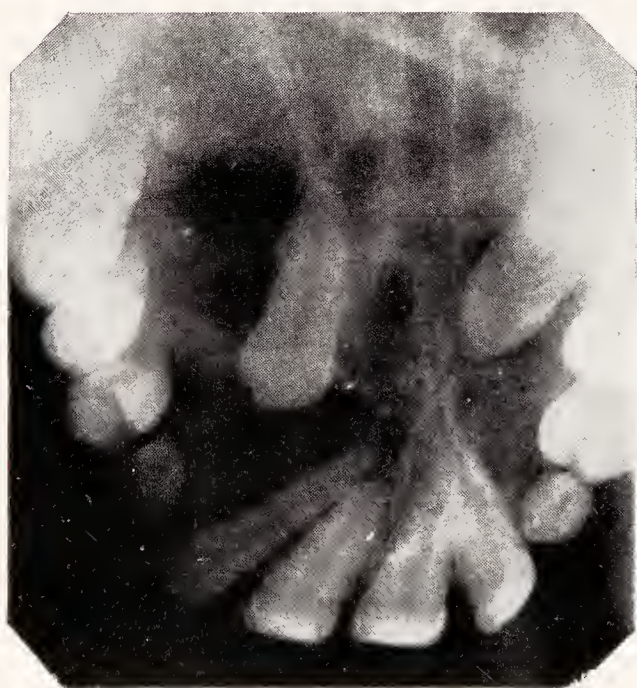


FIG. 378.—Radiograph of a large dentigerous cyst that developed around the crown of an embedded upper left cuspid. (Ennis.)

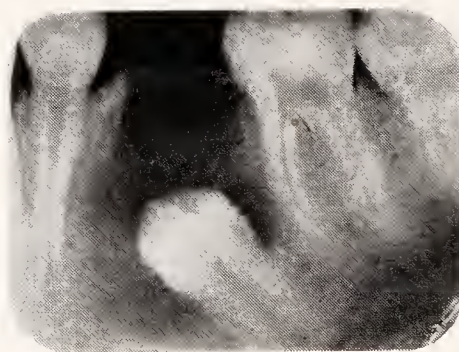


FIG. 379.—Radiograph of a dentigerous cyst that developed around the crown of an embedded lower bicuspid. (Ennis.)

its contents are sterile; only rarely does it become infected. Bacteria may gain access either through a break in the cyst wall, or from an adjacent infectious process.

Dentigerous cysts are more frequently observed in children and adolescents than in older individuals. Their etiology is unknown. Inflammation may play a rôle in those cases in which there is cystic degeneration of the follicle of a permanent tooth germ in the presence of an infected deciduous tooth. Irritation of the tissue overlying the permanent crown may then be the cause of the cystic detachment of the enamel epithelium from the enamel. In other instances,

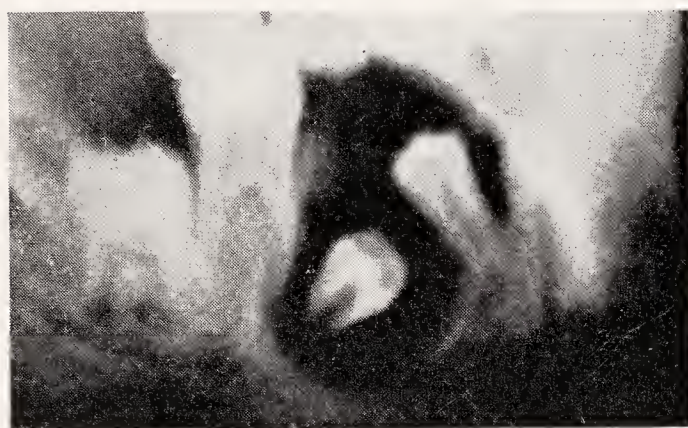


FIG. 380



FIG. 381

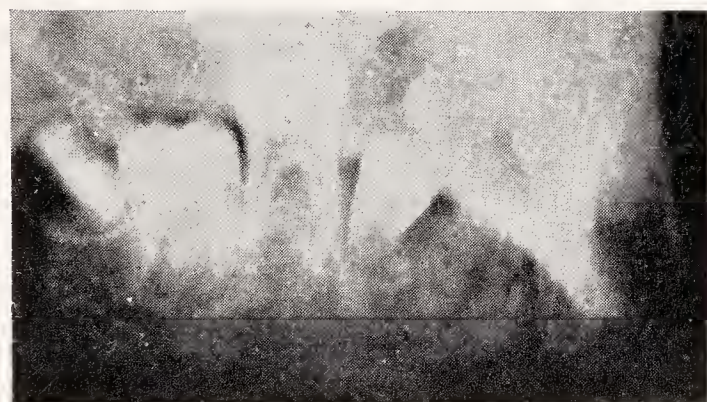


FIG. 382



FIG. 383

FIGS. 380 to 383.—Eruption of tooth following operation of a dentigerous cyst.

FIG. 380.—November 14, 1929. Boy, aged eight years. Dentigerous cyst involving the lower second bicuspid and approaching the first bicuspid and first molar. The operation consisted of the removal of some of the cyst wall near the crest of the ridge and drainage of the amber-colored cyst fluid.

FIG. 381.—January 22, 1930. Advanced formation of the root of the bicuspid. Bone has started to fill in along the walls of the cyst cavity.

FIG. 382.—April 19, 1930. Almost complete regeneration of the bone. Tooth erupting.

FIG. 383.—January 23, 1932. Tooth erupted and in normal alignment. (Puterbaugh and Pike, *The Bur.*)

however, as in the case illustrated in Figure 377, there is no indication of an inflammatory etiology. Perhaps the cystic degeneration of the follicle is caused by a congenital malformation.

The treatment of a dentigerous cyst consists of opening the cyst and keeping it open in order to prevent any further accumulation of fluid in the cavity. New bone regenerates from the inside of the cyst wall and, in time, completely fills the bone cavity. The unerupted

tooth should be removed when there is no possibility of its erupting normally; if, however, the tooth is in such a position that it may come to the surface and occupy its place in the dental arch, it should not be disturbed, for after the pressure of the cyst fluid has subsided, such a tooth often will eventually erupt (Figs. 380 to 383).

MEDIAN ANTERIOR MAXILLARY CYSTS.

Cysts in the anterior maxillary region, located in the midline, have been observed and reported by clinicians and anatomists. They are known as median anterior maxillary cysts (Meyer), incisive canal cysts, or cysts of the nasopalatine duct. They appear as smooth, round sacs with a thin epithelial lining, containing a clear,



FIG. 384



FIG. 385

FIGS. 384 and 385.—Radiographs of median anterior maxillary cysts.

FIG. 384.—Large median cyst with very definite outline of the area of bone destruction.

FIG. 385.—Large cystic cavity in the median line causing bulging of the tissues of the incisive papilla. Considerable destruction of the interdental bone between the central incisors. Both incisors have intact vital pulps.

sterile fluid. Their size varies from that of a small pea to that of a cherry or even larger. The clinical diagnosis is made by the radiograph; since maxillary cysts rarely cause any definite symptoms, they are usually discovered during a routine radiographic examination of the central incisor region. Only seldom do median cysts reach such a size that they cause bulging of the soft tissues in the median line behind the central incisors.

The dentist should be familiar with the existence of such cysts because this knowledge may help to prevent diagnostic errors. In a radiograph of the anterior part of the maxilla, the shadow of a median cyst may overlap the root end of a central incisor and look like a root cyst at the apex of the incisor, although in reality the cystic formation has no connection whatsoever with the tooth (Figs. 384 and 385).

Development of the Palate.—A knowledge of the early development of the palate is necessary to understand the etiology of median anterior maxillary cysts. In a fetus of less than 24 mm. total length, the oral cavity is in open communication with the nasal cavity, no palate having yet been formed. Subsequently the palate develops by a junction of the median primary palate and two lateral secondary palatal plates. The primary palate grows downward from the median nasal process, corresponding to the later premaxilla; the secondary palatal plates develop in a horizontal direction from both sides toward the median line, forming what later becomes the hard and soft palate. When these three parts unite, they form a suture the shape of a Y, the two forks of the Y pointing forward and upward from the median line of the mouth into the nose, the median suture of the hard and soft palate back of the incisive foramen forming the lower part of the Y. The junction point of the forks of this Y corresponds, in the adult, to the oral opening of the incisive canal.

The fusion of the different parts of the palate occurs in embryonic life when the total length of the fetus is between 23 and 28 mm. If this junction fails to take place, either partly or completely, various forms of cleft palate and harelip may result. When the parts of the forming palate come in contact with one another, their epithelial covering disappears to make a solid union possible. Sometimes during this process small islands of epithelium are enclosed between the joining parts. These epithelial remnants are frequently found in children and even in adults along the original fetal Y-shaped line of closure, namely, along the nasopalatine ducts in the incisive canal. Such epithelial islands may undergo cystic degeneration and thus produce median anterior maxillary cysts.

Histology of Median Anterior Maxillary Cysts.—A typical case of a median maxillary cyst was published by the author in 1928. The specimen was the maxilla of a man, aged thirty years, who died of tuberculosis. A radiograph of the central incisors, both of which had intact vital pulps, showed a round, sharply outlined area of bone destruction in the median line between the central incisors. The bone defect was about 8 mm. in diameter and had a punched-out appearance (Fig. 386). The specimen was sectioned in labio-lingual direction. In the area immediately behind the central incisors there is an oblong cyst about 8 mm. in length and 6 mm. in width, occupying the oral portion of the incisive canal and replacing the bone in this region. The wall of the cyst consists of several rows of stratified squamous epithelium (Fig. 387); a layer of loose connective tissue of uniform thickness occupies the space

between cyst and surrounding bone. The bone itself in many places shows evidence of resorption. The cyst apparently was growing at the time of death, and the resulting increase in pressure caused bone resorption. At the lower pole of the cyst, the epithelial lining is continuous with a solid mass of epithelium that extends downward in the direction of the incisive papilla. No evidence of inflammation can be found in the specimen.

The lining of median anterior maxillary cysts consists, in the majority of cases, of stratified squamous epithelium. Occa-

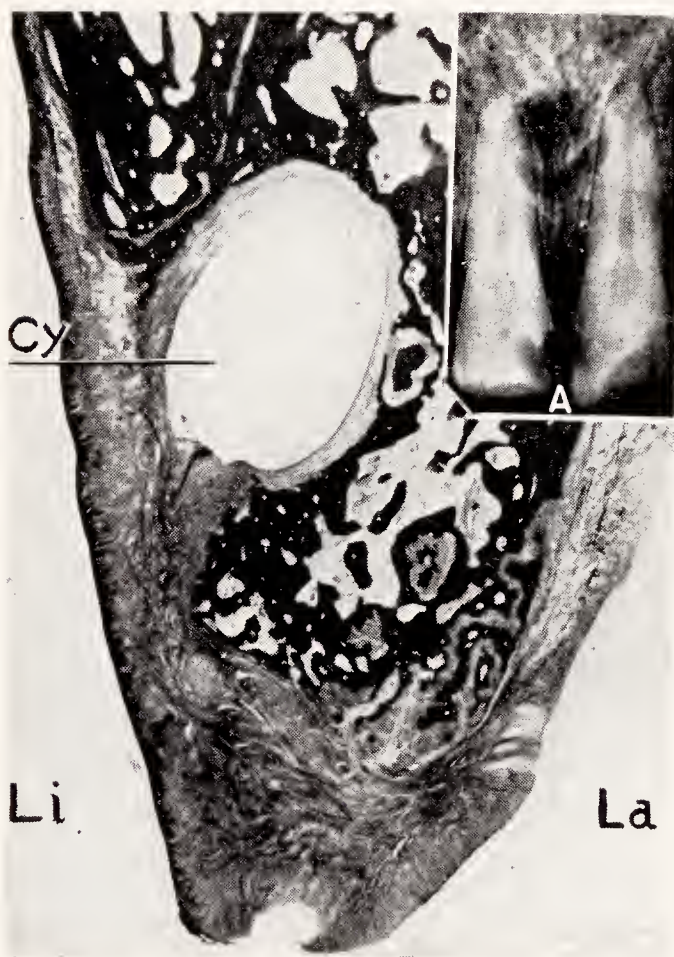


FIG. 386

FIG. 386.—Median anterior maxillary cyst in a jaw specimen. *A*, Radiograph of the specimen showing a round, sharply outlined bone defect between the central incisors. Labio-lingual section through the anterior portion of the maxilla in the median line. *La*, labial side; *Li*, lingual side; *Cy*, cyst of about 6 to 8 mm. in diameter lying near the lower opening of the incisive canal. (Kronfeld, *Korr. f. Zahnärzte*.)

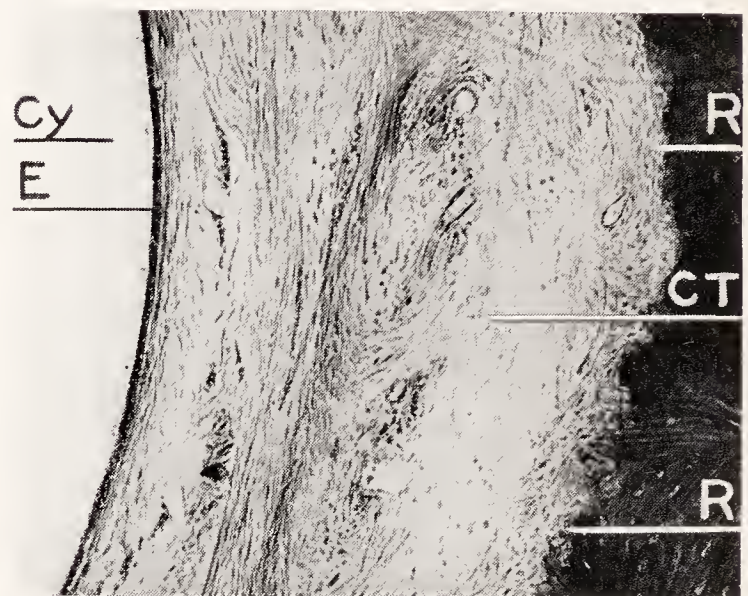


FIG. 387

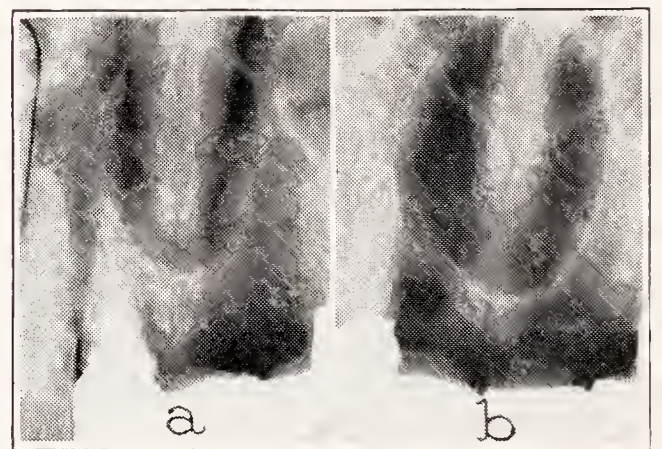


FIG. 388

FIG. 387.—Higher magnification of the cyst wall in Figure 386. *Cy*, cyst cavity; *E*, epithelial lining; *CT*, connective tissue surrounding the cyst; *R*, resorption of the bone. (Kronfeld, *Korr. f. Zahnärzte*.)

FIG. 388.—Two radiographs of a median anterior maxillary cyst; *a*, taken in 1925, and *b*, in 1933. (Stafne, Austin, and Gardner, *Jour. Am. Dent. Assn.*)

sionally, however, cuboidal epithelium and ciliated columnar epithelium are observed, and glandular structures are found in the cyst wall (Stafne, Austin, and Gardner). The columnar epithelium is

probably derived from the nasal end of the nasopalatine duct; the glands are small mucous glands of the type commonly found in the incisive canal.

Median anterior maxillary cysts grow slowly. If radiographs are taken over a period of years, a gradual increase in size can sometimes be noted (Fig. 388). In the case illustrated in Figure 385 the cyst was removed surgically through the palatal surface of the jaw. In the majority of cases no surgical interference is indicated.

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CHAPTER XVIII.

TOOTH FRACTURE.

A TOOTH fracture is a break in the continuity of the dental hard tissues that is caused by a sudden force. Depending upon its etiology, a fracture may be either spontaneous or traumatic: a spontaneous fracture occurs during mastication; a traumatic fracture is caused by an external force (blow, kick, or fall on the face).

PATHOLOGICAL FRACTURES.

Spontaneous or pathological fractures usually occur in teeth that have been weakened in some way, so that the pressure of ordinary mastication is sufficient to cause a break. A frequent cause of such fractures is large fillings with insufficient protection of the cusps or incisive edges. The cusps, together with splinters of dentin, chip off rootward to varying extents. Similarly, a spontaneous cross-fracture of the crown during mastication is sometimes observed in teeth with deep cervical cavities or fillings. Occasionally, also, parts of the crown in intact teeth of old patients with brittle dentin fracture spontaneously. Another cause for pathological fractures is resorptive processes inside the tooth of the type described on page 281. Such internal resorption can undermine and weaken the enamel to such an extent that it suddenly gives way during mastication.

The clinical prognosis in this type of fracture depends upon the amount of root involved. If the fracture involves the crown only, or just a small splinter of the root, it is usually possible to restore the tooth by means of an artificial crown, sometimes by inserting a dowel into the root canal. If, however, the fracture extends farther into the root, an artificial restoration is very difficult.

TRAUMATIC FRACTURES.

Traumatic fractures of anterior teeth are frequently observed as a result of injuries to the teeth and face. Most of them are caused by accidents during play or sports, falls on the face, or automobile accidents (Fig. 389).

From the viewpoint of diagnosis and prognosis, it seems advisable to classify tooth fractures as follows, according to their location and the extent of tooth structure involved:

Fractures located entirely above (crownward of) the epithelial attachment (fractures of the clinical crown only).

Fractures located partly above, partly below, the epithelial attachment (combined fractures of crown and root).

Fractures located entirely below (rootward of) the epithelial attachment (fractures of the clinical root).

The first two forms of fractures can be subdivided into fractures that do and do not expose the pulp.

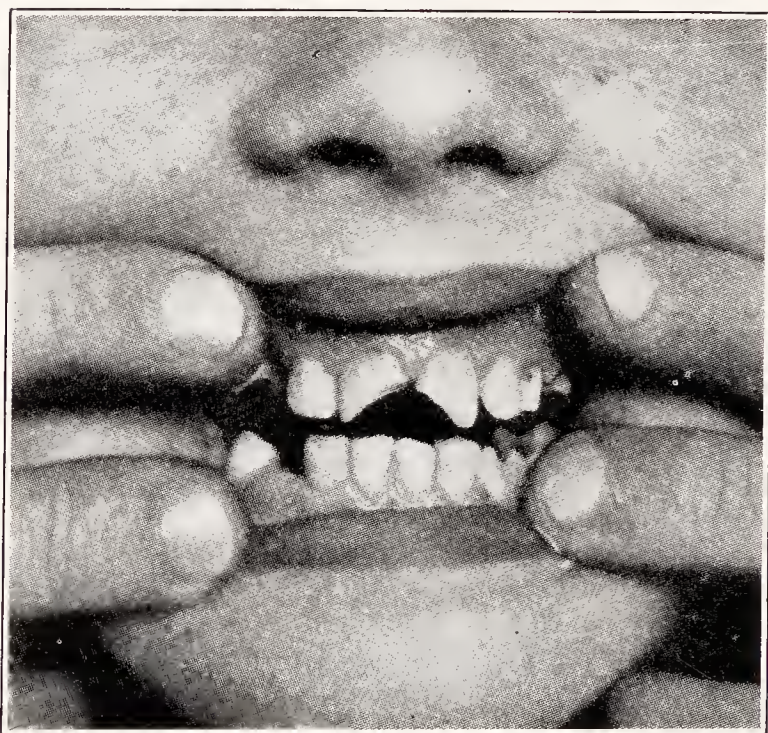


FIG. 389.—Typical sport accident. Fracture of the upper central incisors. (Prinz.)

Fracture of Clinical Crown Not Exposing Pulp.—A traumatic fracture of the crown not exposing the pulp is illustrated in the diagram, Figure 390. Subsequent developments in such a case depend entirely upon the reaction of the pulp. If the pulp is not too close to the exposed dentin surface, secondary dentin will form in the incisal part of the pulp chamber; thus a barricade of gradually increasing thickness is built up between pulp and fractured surface, and the pulp remains vital. Usually the lost part of the tooth can later be replaced by an artificial restoration, such as a porcelain inlay.

The formation of secondary dentin under a fracture of part of the crown has been studied experimentally. Figure 391 shows a lower incisor of a dog, the tip of which was broken off eight weeks previous to the removal of the specimen. A considerable amount of secondary dentin has been formed in the pulp chamber.

If, in a fracture of this type, the pulp is very close to the exposed dentin surface, bacteria usually gain access to the pulp through the thin layer of dentin before the pulp tissue can protect itself by the formation of secondary dentin. Pulpitis develops, and the pulp is lost. Then the tooth has to be treated as though the pulp had been exposed.



FIG. 390

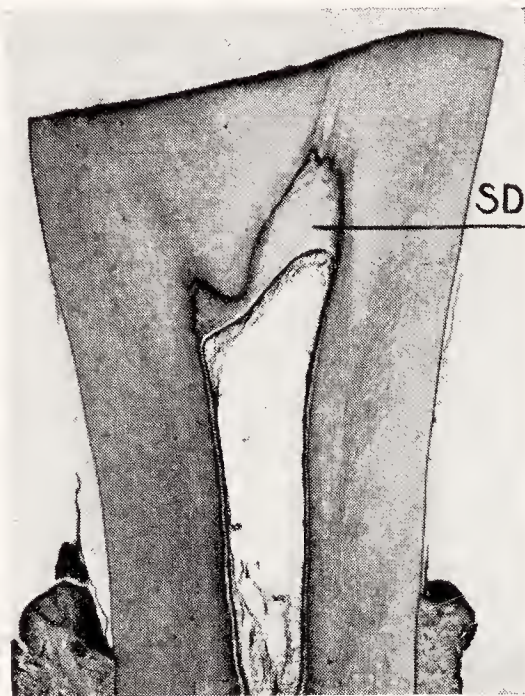


FIG. 391



FIG. 392

FIG. 390.—Diagram of a traumatic fracture of an upper incisor. The pulp is not exposed by this fracture.

FIG. 391.—Traumatic fracture of a dog's incisor produced experimentally eight weeks before the animal was killed. The pulp was not exposed by this fracture. *SD*, secondary dentin formation, corresponding to the extent and location of the fracture. (Courtesy of E. D. Coolidge.)

FIG. 392.—Diagram of a traumatic fracture of an upper incisor with exposure of the pulp.

In every tooth fracture without pulp exposure, it is possible that the blow that fractured the crown may also have severed the pulp vessels at the apical foramen, thus causing necrosis of the pulp. Therefore, it is always advisable to test the vitality of the pulp at frequent intervals following the fracture, in order to make sure that the pulp has remained vital.

Fracture of Clinical Crown Exposing Pulp.—If a large piece of the crown is broken off by a blow or fall, the pulp, as a rule, is exposed (Fig. 392). Then the pulp must be removed and the root canal filled. An artificial restoration of the lost part of the crown can be made, using the root canal for anchorage.

The possibility that a traumatic pulp exposure may heal by the deposition of dentin that would seal the opening is theoretically possible. But usually when such a procedure of pulp capping is attempted, the pulp dies sooner or later and has to be removed.

Fracture Located Partly Above, Partly Below, Epithelial Attachment (Combined Fracture of Crown and Root).—Most of the combined fractures of crown and root are spontaneous fractures of posterior teeth with large fillings. The pulp is almost always involved in fractures of this type (Fig. 393); however, in older people in whom the size of the pulp chamber is greatly reduced, it is possible that a cusp together with a splinter of crown and root may be broken off without exposing the pulp.

Fracture Located Entirely Below Epithelial Attachment (Fracture of Clinical Root).—If the crown of a tooth is struck, the enamel of the crown frequently remains intact, whereas the root, being more delicate, is fractured; the thinner apical portion of the root seems to be especially liable to be broken at such a time. Complete cross-fractures and incomplete fractures of the root, including cracks and tears in the root surface, will be considered separately. The former type is usually visible in the radiograph; the latter is found only when the tooth is examined microscopically.

COMPLETE CROSS-FRACTURE OF ROOT.

In complete cross-fracture of the root, the tooth is usually sore and loose, but only the radiograph can make the diagnosis of a fracture a certainty. The line of fracture runs straight across or obliquely through the root (Fig. 394). If the fracture occurs near the neck of the tooth, the crown may be quite loose; if the apical third or quarter of the root is broken, the loosening is sometimes insignificant (Fig. 395).

When examining a traumatic tooth fracture clinically, one must keep in mind the possibility of a multiple fracture. If, for instance, in an accident part of the crown of an incisor is broken off, there is always the possibility of a cross-fracture of the root in addition to the fracture of the crown; therefore, a radiographic examination is necessary before the diagnosis can be considered complete.

Only in fractures of the root is healing in a biological sense possible. In fractures that involve the clinical crown, there can be no reparative processes whatsoever on the fractured surfaces. In those that are located partly above and partly below the epithelial attachment, healing of the fractured portion below the epithelial attachment is possible, but the extension of the fracture into the clinical crown usually leads to infection of the deeper tissues and prevents healing. Only when the entire line of fracture is well inside the epithelial attachment does no infection occur, and only

then is healing possible. The final outcome of such a root fracture is governed by the same fundamentals as the healing of a simple bone fracture.

The end-result of any bone fracture is either solid union by newly formed hard substance, or a fibrous union without bony connection, or infection and suppuration, which make healing difficult or impossible. From a clinical viewpoint the first of these three possibilities is obviously the most desirable. Four conditions are essential for such a solid bony union of a fracture: (1) the fragments must be in close adaptation, (2) they must be immobilized in this position, (3) infection must be absent, and (4) the patient's general health

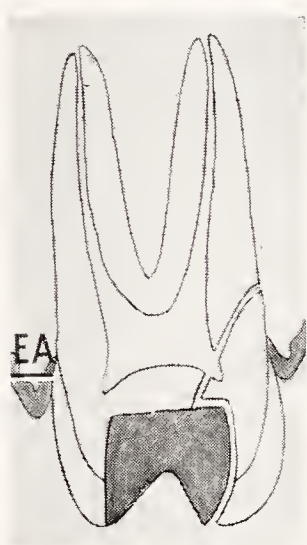


FIG. 393



FIG. 394

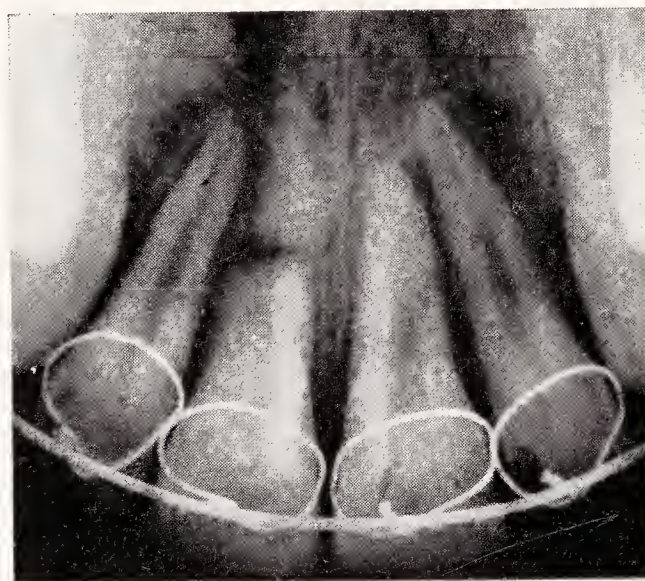


FIG. 395

FIG. 393.—Diagram of spontaneous fracture of an upper bicuspid with a large filling. Exposure of the pulp. *EA*, position of the epithelial attachment on the tooth surface. The fracture is located partly above (crownward of) the epithelial attachment, partly below (rootward of) the epithelial attachment.

FIG. 394.—Diagram of a traumatic cross-fracture in the apical third of the root of an upper incisor.

FIG. 395.—Radiograph of a cross-fracture of the root of an upper incisor. Considerable displacement of the fragments. Fixation by wiring.

must be such as to make reparative and regenerative processes possible. The following application of these conditions can be made to root fractures:

1. In a typical bone fracture, union is possible only if the fragments are in contact or at least very close together. The healing of a fracture is initiated by a proliferation of the osteogenic cells of the periosteum and endosteum; these cells differentiate into osteoblasts that lay down bone matrix. By subsequent calcification of this matrix, a solid bony callus is formed between the fragments. If the distance between the fractured ends is too great, the callus cannot bridge the gap; instead, each end is covered separately by

new bone, and the ends are united merely by fibrous tissue. This is known as pseudarthrosis.

The same is true of a cross-fracture of the root. Shortly after the accident, the hemorrhage that invariably takes place within the line of fracture is resorbed or organized by ingrowing fibroblasts and cementoblasts. First, a fibrous bridge is formed between the fragments; later on, cementum is deposited along the line of fracture. If the root fragments are in close adaptation, they are cemented together by the cementum, and the continuity of the root is restored. If the fractured ends are farther apart, each is covered separately by cementum. Fibrous tissue remains between them, and no union takes place. Healing of this type may be entirely satisfactory for the maintenance of function, providing that the coronal fragment is long enough to insure sufficient periodontal attachment.

2. In a bone fracture a solid union can be expected only if the fractured ends are immobilized, hence the general use of splints and plaster casts in the treatment of fractures. The same is true of a root fracture. If the coronal fragment is loosened by the trauma and in addition is subjected to stress during mastication, a solid union cannot be expected. Therefore, care should be taken to give a fractured root a chance to heal by immobilizing the coronal fragment as much as possible. This can be done in several ways. A splint may be constructed from an orthodontic arch wire to which the fractured tooth is ligated, or bands may be made for the fractured tooth and its neighbors, which are soldered together and cemented on the teeth for a period of several months. In addition to the construction of a splint it is sometimes necessary to shorten the fractured tooth or its antagonists by grinding. The process of repair can be followed in radiographs taken at intervals; the line of fracture becomes more and more indistinct until finally it disappears entirely, indicating that calcified tissue has united the fractured ends.

3. The healing of a bone fracture is greatly delayed and sometimes entirely prevented by infection and inflammation. This is equally true of a root fracture. For this reason the condition of the root canal has to be considered very carefully. If, at the time of fracture, the tooth was pulpless and had an infected, poorly treated, or untreated root canal, the possibility of retaining the tooth is practically *nil*. The infection invariably spreads from the root canal or from the infected periapical region into the traumatized tissue near the line of fracture; this makes repair impossible and leads to additional bone destruction around the root. The same happens if the line of fracture is located close to the bottom of the gingival crevice.

Infection then reaches the fracture line and prevents healing of the fragments.

4. In some patients bone fractures do not heal satisfactorily despite all therapeutic measures. In such people the general reparative power of the organism is below par because of old age, poor health, malnutrition, or some other cause. The same holds true of tooth fractures; in young, healthy individuals, satisfactory tissue repair can usually be expected; in older or in poorly nourished patients, the chances are correspondingly lessened.

Austin, in a report from the Mayo Clinic, found that among 40 single-rooted anterior teeth with cross-fractures of the roots 31 responded to the vitality test. This high percentage of teeth in which the pulp remained vital following fracture is indicative of the relatively high resistance of the dental pulp to traumatic injuries, providing infection has not gained access to it.

Histological Findings in Fractured Roots.—The reparative and regenerative changes in several cases of root fracture have been reported in dental literature. Gottlieb described a fracture of the apical half of the root of an upper cuspid of a man who died at the age of twenty-two years. The crown was intact; nothing was known about the date of the accident or the type of trauma that had caused the fracture. The pulp of the tooth had remained vital, although the root was broken into several large and small pieces; bone and cementum had been deposited over all of the area of fracture, covering the exposed dentin and partly uniting the fragments. Bone and cementum were also found in the pulp canal. Although it undoubtedly took a great force to cause such splintering of the root, the pulp tissue remained vital, a good example of the high resistance and reparative power of the pulp.

Howe reported the histological findings in a cuspid that had sustained a cross-fracture of the root six months before the extraction. A solid union of the fragments had taken place. The newly formed hard substance between the fragments originated from both periodontal membrane and pulp; it consisted of cementum and irregular dentin.

Boulger reported a case of cross-fracture of the roots of the lower central incisors. The cause was known to have been an accident (fall on the face) thirty-three years previous to the time of removal of the specimen. Immediately following the accident the teeth were sore and loose, but they became firm again without any treatment. The apices did not unite with the upper part of the roots, but remained embedded in the jaw; the distance between the fragments was con-

siderable, probably because of an occlusal movement of the upper fragments during the thirty-three years following the accident. Figure 396 shows the roots of the central incisors and the apex of the left central incisor. In a higher magnification of the root end of the right incisor, it can be seen that the fractured surface is completely covered by cementum (Fig. 397); the pulp is vital in the upper as well as in the apical fragments. The apical fragments are completely



FIG. 396.—Fracture of the root ends of the lower central incisors. Thirty-three years previous to the removal of the specimen the patient, a woman aged forty-five years, fell on her face and fractured the central incisors. Radiographically, the roots of both central incisors show cross-fractures. The left apex lies close to the upper fragment; the right apex is located at a considerably deeper level. The pulps of both teeth remained vital. The histological specimen shows the apical fragment of the left incisor separated from the upper portion of the root by connective tissue. *CT*, connective tissue between the fragments of the left incisor; *C*, deposits of cementum upon the fractured dentin surfaces; *PM*, periodontal membrane surrounding the fragments. (Boulger, Jour. Am. Dent. Assn.)

surrounded by cementum; they contain thin strands of living tissue that are in connection with the periodontal membrane at the upper, fractured surfaces and at the apex of the fragments.

The author described a cross-fracture of an upper central incisor in the cervical one-third of the root. The pulp remained vital; the two fragments did not become united, but both dentin surfaces were covered by cementum, and a periodontal membrane extended from

one fragment to the other. A large amount of irregular dentin had been formed in the pulp chamber and along the root canal (Fig. 398).

In the reparative processes following root fracture, the deposition of cementum upon the fractured surface is often preceded by some resorption of the exposed dentin surface (see Fig. 397). This preliminary resorption is probably the expression of some tissue injury caused by the trauma; the injured soft and hard tissues must be eliminated before repair can take place.

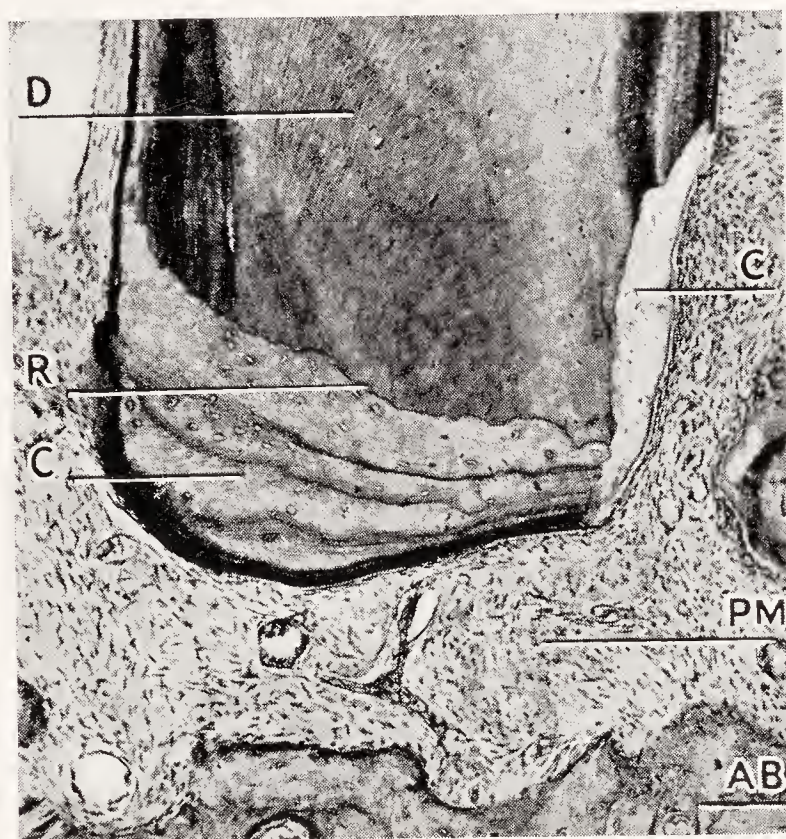


FIG. 397.—High magnification of the root end of the right central incisor of Fig. 396. *D*, dentin; *R*, line of resorption on the dentin surface; *C*, deposits of cementum on the resorbed dentin surface; *PM*, periodontal membrane; *AB* alveolar bone. (Boulger, Jour. Am. Dent. Assn.)

Incomplete Fracture of Root.—Fractures of small portions of the root are observed in the form of cracks or tears in the cementum. They have no clinical significance and cannot be recognized by means of the radiograph; however, such small traumatic defects on the root surface are frequently found in the microscopic examination of teeth and contribute to our knowledge of the reparative power of cementum and periodontal membrane.

The author described a specimen of a lower incisor in which part of the cementum had been torn away from the root (Fig. 399). This kind of fracture originates in the following way: When a tooth is struck suddenly, a piece of cementum, which is more firmly attached to the fibers of the periodontal membrane than to the dentin, may be torn from the root surface. The cementum splinter lodges in the periodontal membrane. Figure 399 shows the tear extending over

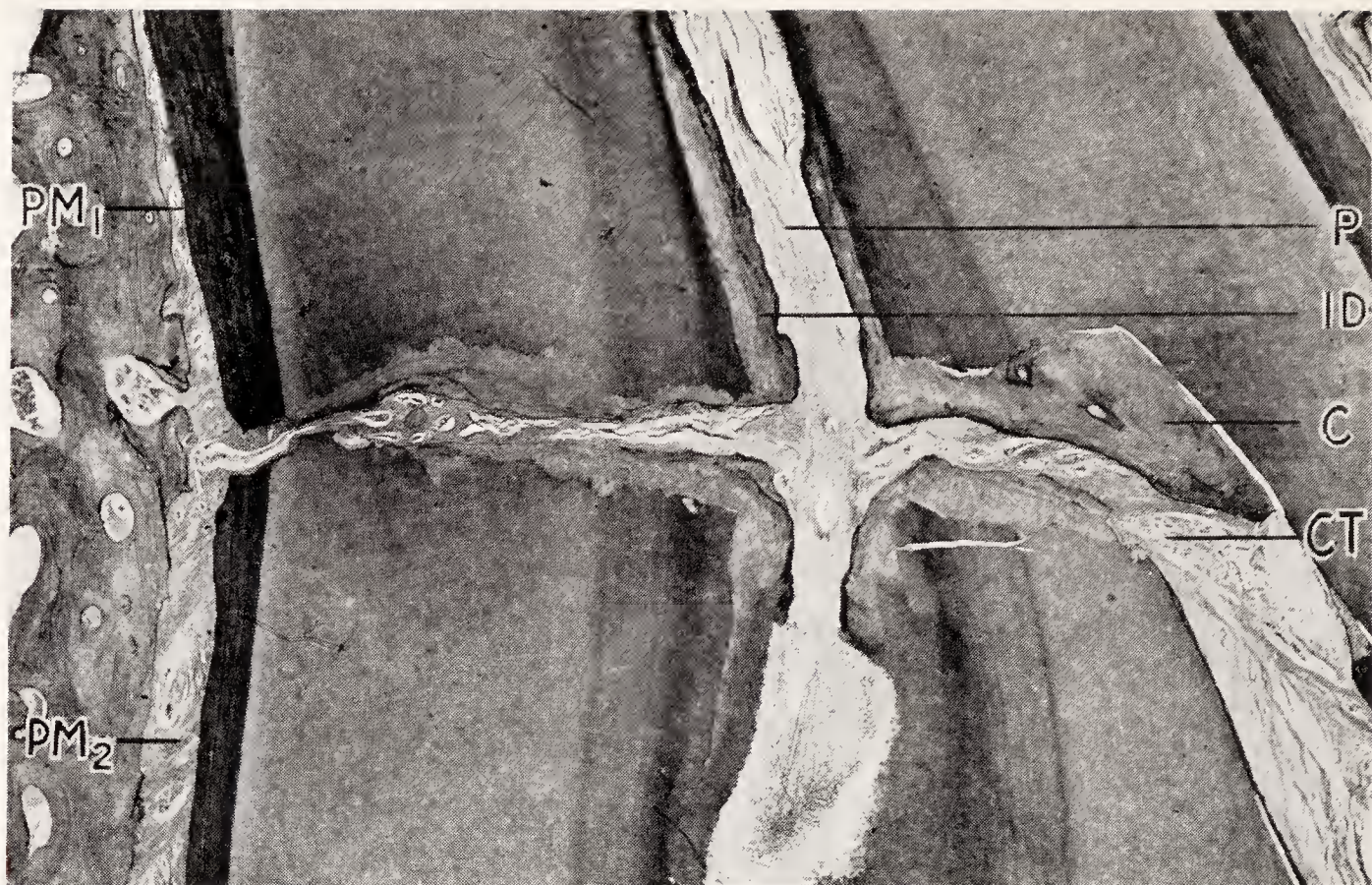


FIG. 398.—Cross-fracture of the root of an upper central incisor. The photomicrograph shows the fracture line at the point where the pulp tissue crosses it. *P*, pulp; *ID*, irregular dentin; *C*, cementum lining fracture; *CT*, connective tissue between fragments; *PM*₁, thin periodontal membrane of apical fragment; *PM*₂, thick periodontal membrane of incisal fragment. (Kronfeld, Jour. Dent. Res.)

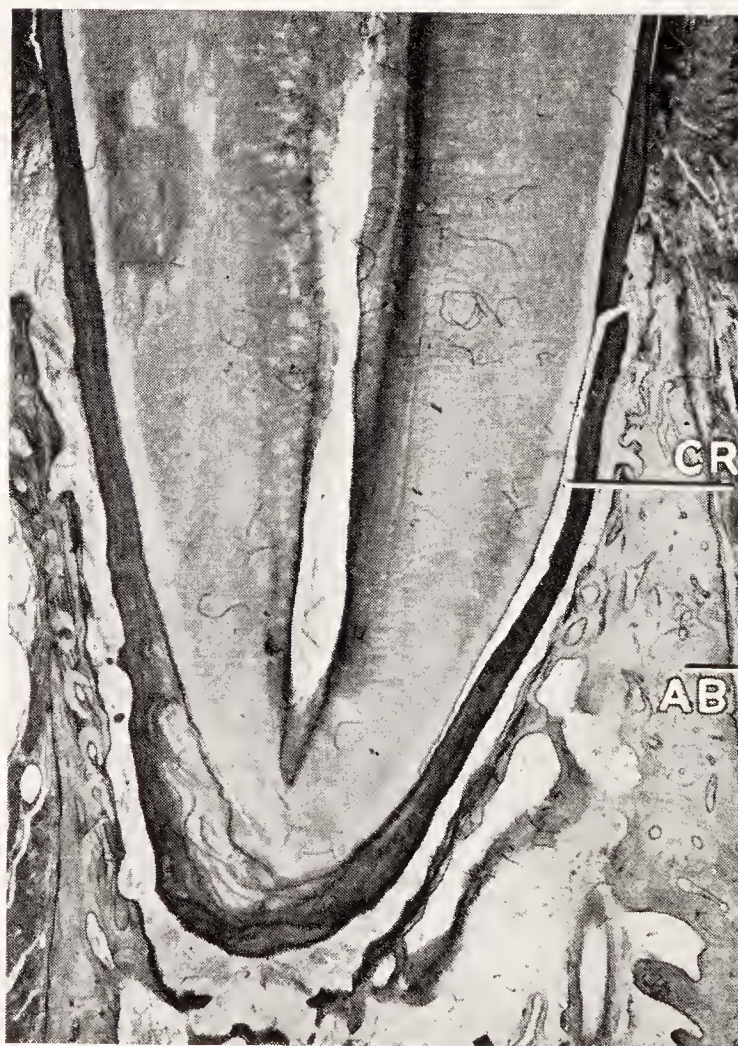


FIG. 399.—Traumatic separation of cementum from the dentin surface. Labio-lingual section through lower central incisor root. *CR*, crack between cementum and dentin on lingual side of root; *AB*, alveolar bone. (Kronfeld, Jour. Dent. Res.)

more than one-half of the clinical root. The upper part of the crack is filled with ingrowing connective tissue from the periodontal membrane. The apical part contains tissue fluid and a fine network of fibroblasts and wandering cells. These cells represent the first step toward the organization of the fluid that entered the crack immediately following the trauma; as the fibroblasts grow and multiply they form connective tissue on the walls of the crack and finally cementum, thus reuniting the fractured parts (Fig. 400).

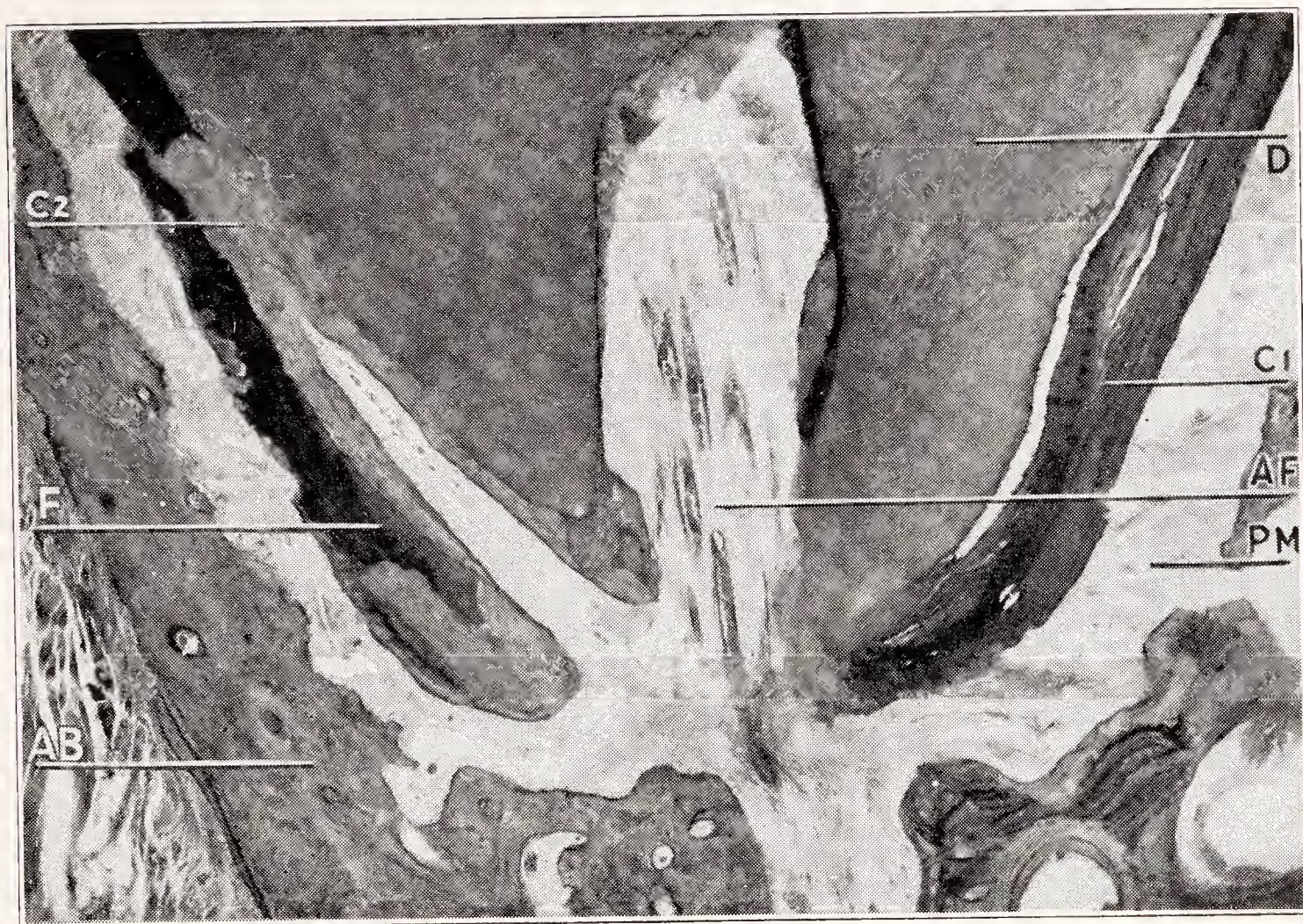


FIG. 400.—Apex of lower right lateral incisor showing result of excessive occlusal stress. A portion of the cementum at the apex has been torn loose from the dentin surface. *D*, dentin; *AB*, alveolar bone; *PM*, periodontal membrane; *C*₁, old cementum; *C*₂, new cementum; *F*, fractured cementum partially repaired; *AF*, apical foramen. (Coolidge, Jour. Am. Dent. Assn.)

Figure 401 represents a similar fracture of longer standing. Here a splinter of cementum and dentin had been torn loose from the root near the alveolar margin; this fragment, as well as the corresponding defect on the root surface, has been completely covered by deposits of cementum.

These findings suggest the following order of tissue changes after a tooth fracture: Immediately after the injury, blood and tissue fluid fill the space between the fragments. From the surrounding periodontal and pulp tissue, fibroblasts and wandering cells prolifer-

ate into the crack; the fibroblasts form connective tissue. Resorption may or may not take place on the fractured ends; if it does take place cementum is deposited later upon an uneven, eroded surface; if resorption does not take place, cementum is laid down upon the original surfaces of the fracture. If the fragments are adjacent, cementum may tie them firmly together; if they are farther apart, each fragment may be covered separately, and a periodontal space may remain between them.

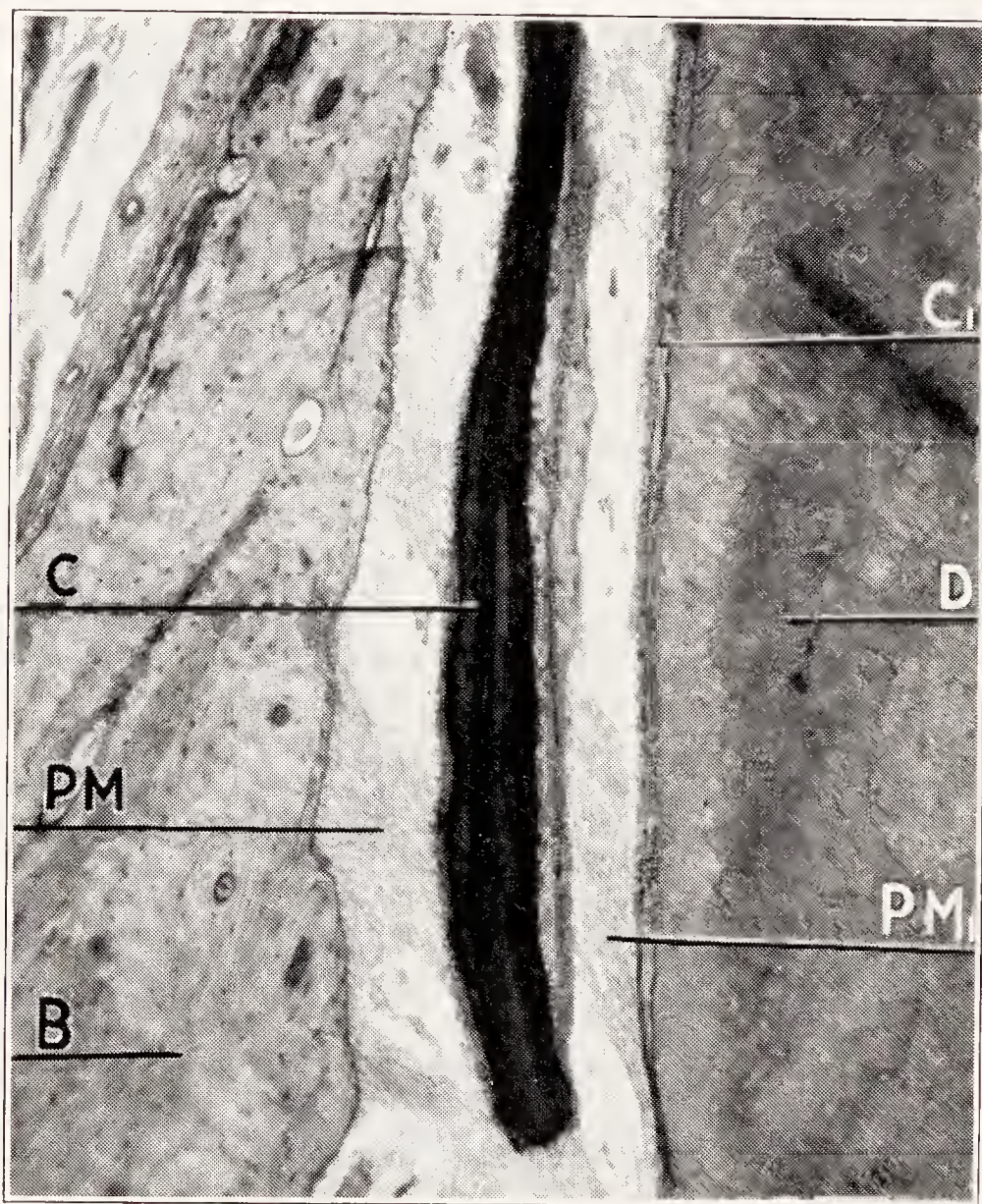


FIG. 401.—Traumatic cementum injury. A splinter of cementum (*C*) has been torn away from the root surface and is suspended in the periodontal membrane. The surfaces of the splinter and the dentin surface are covered by new cementum. *D*, dentin; *C*₁, new cementum on the dentin surface; *PM*₁, periodontal membrane fibers between root and splinter; *PM*, periodontal membrane; *B*, alveolar bone. (Kronfeld, Jour. Am. Dent. Assn.)

The pulp often remains alive, especially in young teeth with wide pulp canals and highly vital tissue. A collateral blood supply to the pulp develops from the periodontal vessels (Fig. 398). Sometimes, though, the pulp dies. Clinically, the fate of the pulp in a

tooth with a fractured root can be decided only by close observation and repeated vitality tests in the weeks and months following the accident.

Pulp Changes in Fractured Teeth.—A possible sequel to traumatic injury to a tooth, either with or without fracture, is calcific degeneration of the pulp tissue and, finally, solid calcification of the pulp cavity (Fischer, Wenzel). The radiographic examination of a tooth several years after a trauma may reveal that the pulp chamber has been obliterated. Histologically, the calcified material may show the characteristics of secondary dentin, or, as a result of metaplasia of the pulp, it may be cementum or a bone-like tissue.



FIG. 402

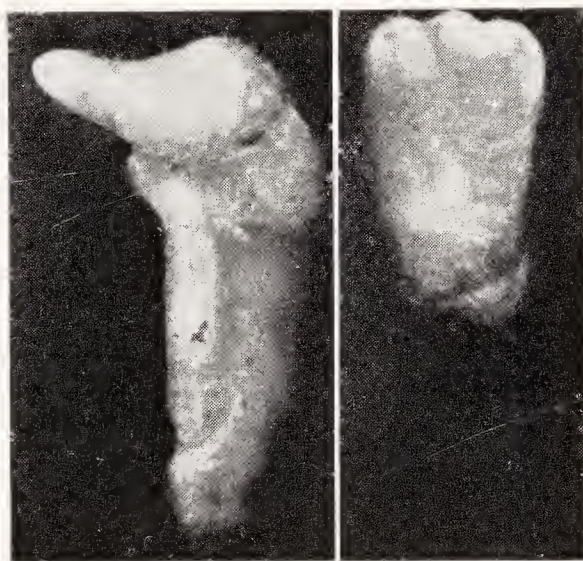


FIG. 403

FIGS. 402 and 403.—Dilaceration of a lower permanent central incisor caused by a traumatic injury to the deciduous predecessor. (Courtesy of John Y. Ing.)

FIG. 402.—Radiograph of the dilacerated tooth.

FIG. 403.—The extracted incisor seen from the labial and occlusal sides. The crown is attached to the root at an angle of about 90 degrees; the incisal edge points labially. In a view of the tooth from the occlusal side, the lingual surface of the crown is visible.

PARTIAL FRACTURE (DILACERATION).

Dilaceration is a deformity that consists of a bend or crease at the junction between crown and root. In the lower central incisor illustrated in Figures 402 and 403, the root was in normal position in the jaw, but the crown was attached to the root at an angle of approximately 90 degrees, the incisal edge pointing toward the labial side. Figure 404 shows a ground section through a similar tooth.

The cause for dilaceration is a pre-eruptive injury, usually a blow or fall, which severely jars the deciduous tooth and is transmitted to the crown of the underlying permanent tooth germ. The crown of the latter is partly detached from the dental papilla, and the

formation of the root continues at an angle to the axis of the crown. In the case shown in Figures 402 and 403, the patient fell downstairs at an early age, during which accident a deciduous lower central incisor was forced out of its socket. The permanent incisor germ apparently was injured in the same accident, for when it erupted several years later it was deformed.



FIG. 404.—Longitudinal ground section through an incisor with dilaceration.
(Burchard and Inglis.)

ROOT ENDS REMAINING IN JAW AFTER EXTRACTION OF TEETH.

If a piece of a root is fractured during extraction and left in the jaw, the outcome varies according to the condition of the fragment at the time of the operation. If the root end was chronically infected, three things are possible: (1) The root fragment may be gradually eliminated. It may work its way to the jaw surface and finally be lost. (2) The root fragment may remain in the jaw. The tissues may heal over it and form a sinus leading from the fragment to the surface, through which a purulent exudate is discharged. (3) The jaw tissues may heal completely over the fragment, the latter remain-

ing in the jaw without any clinical symptoms, as so-called residual infection.

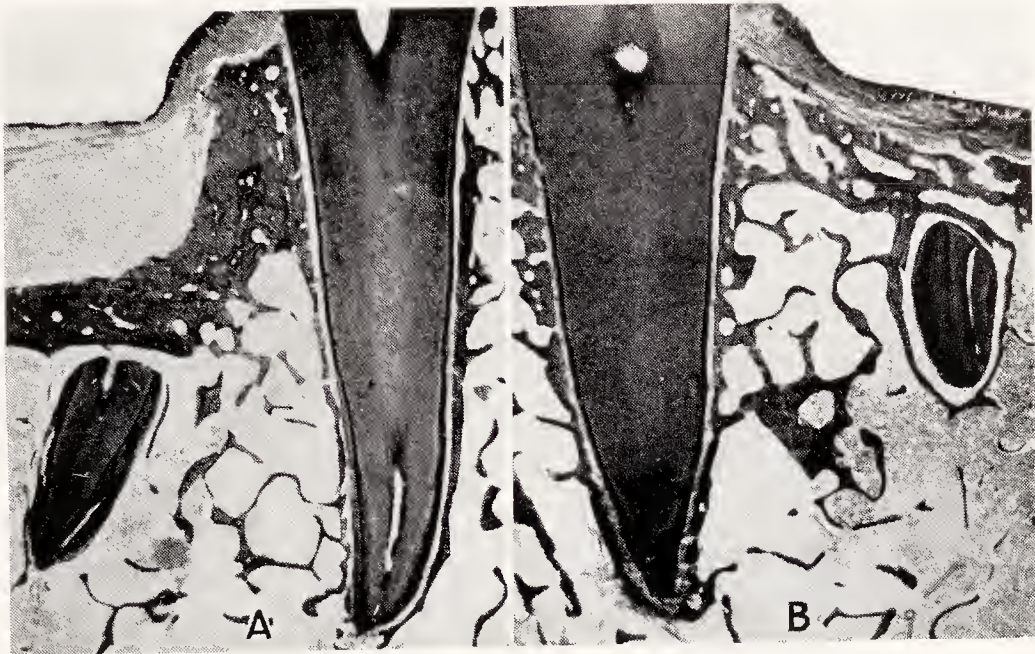


FIG. 405.—Right and left side of the mandible of a woman, aged thirty-eight years. Mesio-distal sections through the region of the second bicuspid and first molar. The mandible was edentulous on both sides posterior to the second bicuspid. Histological examination revealed on both sides the fractured apex of the mesial root of the first molar that had remained in the jaw. Both of these fragments are embedded in the surrounding tissues without inflammatory reaction. (Kronfeld, *Ztschr. f. Stomatol.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)



FIG. 406.—High magnification of the surface of the fragment in Figure 405, B. D, dentin; P, pulp tissue in the root canal; C, cementum deposited upon the fractured dentin surface; FM, fat marrow surrounding the fragment; AB, alveolar bone. (Kronfeld, *Ztschr. f. Stom.*, courtesy of Urban and Schwarzenberg, Berlin-Wien.)

If the root was not infected, that is, if the extracted tooth had an intact or only superficially inflamed pulp, a fractured apex may heal

without any inflammatory reaction: the alveolar bone and soft tissue completely regenerate, the pulp stump in the fragment remains alive, and the fractured surface is covered by cementum. The author described the tissue changes around the fractured ends of the mesial roots of the lower first molars on both sides of the same jaw (Fig. 405); both fragments were completely surrounded by bone and showed no inflammatory changes (Fig. 406). Euler, Szabo, Thomas, and Zemsky have reported deposits of cementum on the fractured surfaces of roots left in the jaw after extractions.

Although favorable conditions are occasionally found around embedded root ends, it must not be inferred that any carelessness in removing fractured root apices is justified. If the field of operation is infected, or if infection gains access to the fragment during the extraction, the remaining root end is a potential source of danger. The old rule still stands: An extraction is not completed unless the entire tooth is removed.

LUXATIONS OF TEETH.

In a luxation a tooth is either partially or completely severed from its socket by a trauma. In a partial luxation the tooth is sore, loose, and often protrudes from its socket. The therapy is the same as in a fracture: reposition and immobilization (splint) until the tooth becomes firm. A radiograph should always be taken in order to determine whether or not the root is fractured.

After a tooth has healed following luxation, the vitality of its pulp should be tested, as this organ often suffers apical hemorrhage and lacerations and is likely to become necrotic. If the pulp does not respond to the electrical and thermal vitality tests, the tooth should be opened, the pulp removed, and the root canal filled. In complete luxation the therapy consists of replanting the knocked-out tooth. Usually such replanted teeth remain in the jaw and function well for a while; but eventually they are resorbed and are lost (see page 272). Before replantation it is necessary to remove the pulp tissue and to fill the canal, as otherwise the pulp would decompose and cause inflammation around the replanted tooth.

Traumatic fractures and luxations of teeth are often accompanied by fractures of small parts of the alveolar process surrounding these teeth. Bone fractures of this type usually heal promptly if the teeth are properly held in place by a splint.

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CHAPTER XIX.

HEALING OF EXTRACTION WOUNDS. HISTOLOGY OF EDENTULOUS JAWS.

HEALING OF EXTRACTION WOUNDS.

SEVERAL tissues play a rôle in the healing processes following the extraction of teeth. The epithelium covers the wound and restores the continuity of the epithelial covering of the oral cavity. The subepithelial connective tissue of the alveolar process proliferates and forms the scar tissue in the wound. The periosteum and the endosteum of the alveolar process furnish new bone and fill the space formerly occupied by the root. All of these changes have been studied experimentally in animals; these experimental findings, in turn, have recently been corroborated by the histological examination of human jaws from which teeth had been extracted shortly before death.

In American dental literature, histological studies of the healing of extraction wounds were first reported by Schram. More extensive experimental work was later done by Claflin. In two reports, which also include a complete survey of the foreign literature on the subject (Euler, W. Meyer, Balogh, H. Meyer, and others), Claflin described the tissue changes associated with the healing of simple extraction wounds, infected extraction wounds (dry socket), and experimental alveolectomies.

UNDISTURBED HEALING OF EXTRACTION WOUNDS.

Immediately following the extraction of a tooth, a blood clot fills the empty socket. The clot consists of a net of fibrin threads the meshes of which are filled with red and white blood cells. The gingival tissues, which have lost their support, collapse over the wound and form a protective covering for the clot. In the next few days the clot undergoes a process of organization. The fibroblasts in its periphery divide, and new fibroblasts grow into the coagulated blood. This is shown in Figure 407, a three-day old extraction wound of a dog's premolar, which is completely filled with the clot. A higher magnification reveals the presence of young fibroblasts with

star-shaped extensions between the red blood cells in the periphery of the clot. By the fifth or sixth day, the epithelium has grown from the periphery across the surface of the clot and completely covers the wound. Shortly afterward, on the seventh day, the first evidence of bone formation is found along the walls of the socket. This bone is laid down in delicate trabeculae, which come from the original alveolar bone and proliferate into the organized clot (Fig. 408). Simultaneous with the bone formation in the socket is a process of

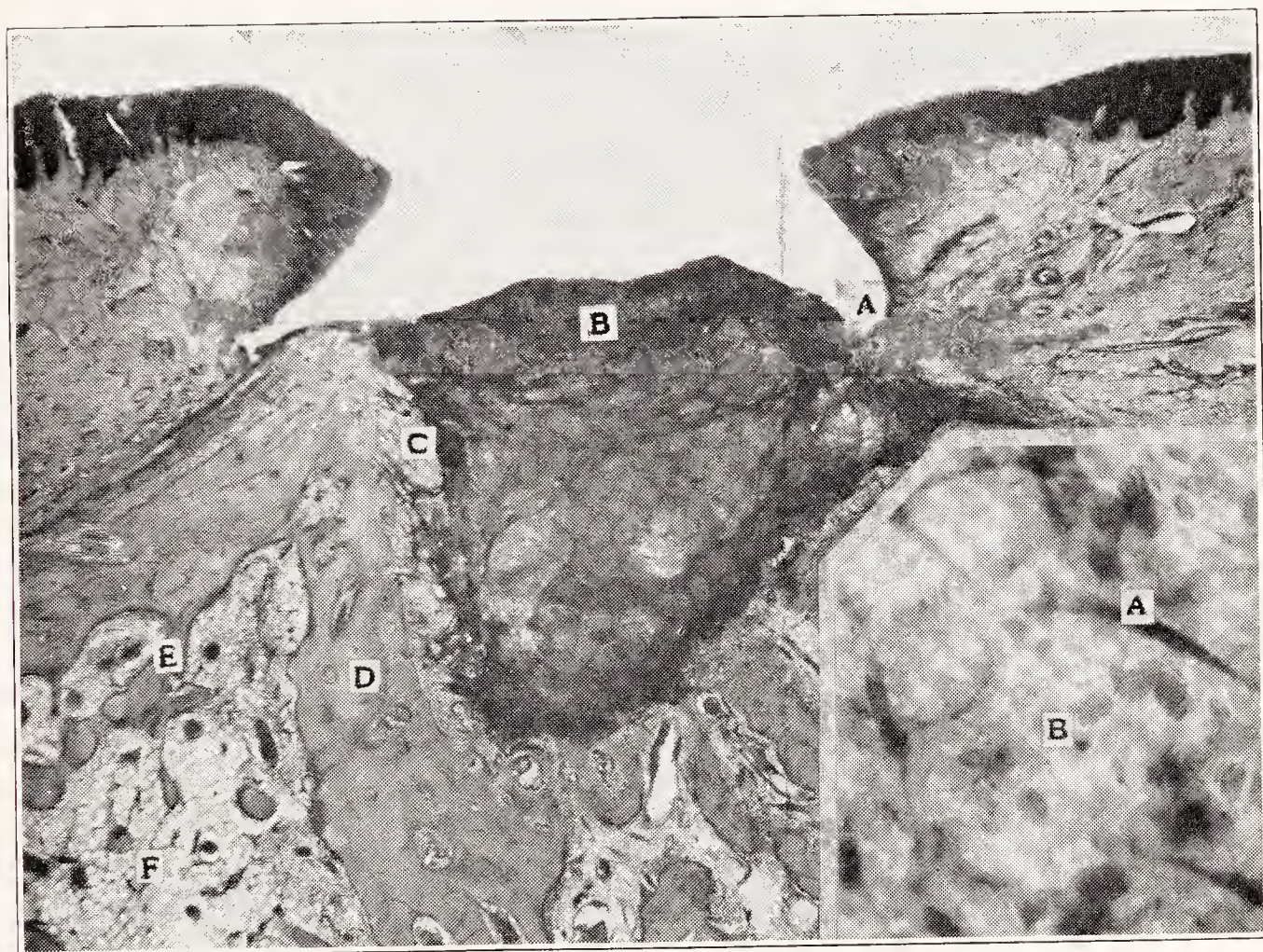


FIG. 407.—Extraction wound, three days old, of a first premolar of a dog. *A*, proliferation of epithelium; *B*, fibrin network over clot; *C*, remnants of periodontal membrane; *D*, cribriform plate of alveolar bone; *E*, supporting osseous structure; *F*, bone marrow. Insert: *A*, fibroblasts; *B*, red blood cells. (Claffin, Jour. Am. Dent. Assn.)

bone resorption along the crest of the alveolus; as a result, the socket is being filled with new bone at the same time that its depth is being decreased, until the newly formed bone has reached the level of the resorbed crest. At first the original cribriform plate can be easily recognized; later, however, there are extensive resorptive changes in the alveolar wall, by which the latter is eventually assimilated into the trabecular system of the edentulous jaw area.

Claffin found in young dogs that the sockets are completely filled with bone approximately thirty days after the extraction. In man

analogous tissue changes take place, but more slowly; in the dog the various reparative changes occur about twice as fast as the corresponding changes in man.

The extraction wound of a human lower molar is shown in Fig. 409. The upper portion of the socket contains fibrous connective tissue;



FIG. 408.—Extraction wound, eleven days old, of a first premolar of a dog. *A*, epithelium covering extraction wound; *B*, subepithelial connective tissue; *C*, blood clot; *D*, new bone formation; *E*, cribriform plate; *F*, bone marrow. (Claffin, Jour. Am. Dent. Assn.)

near the fundus remnants of the original blood clot are still visible. Delicate trabeculæ of new bone were growing from the fundus of the alveolus. The alveolar crest and the crest of the interdental septum are densely covered with osteoclasts. In this way the socket was being filled with bone and at the same time its depth was being reduced.

DISTURBED HEALING OF EXTRACTION WOUNDS.

The changes described so far take place if the wound is allowed to heal without disturbance. Occasionally, however, complications arise; these are clinically known as dry socket. As indicated by the name, the alveolus, two or three days after the extraction, is empty or contains débris. The blood clot has disintegrated and the bony walls of the socket are exposed, giving the wound a “dry” appear-

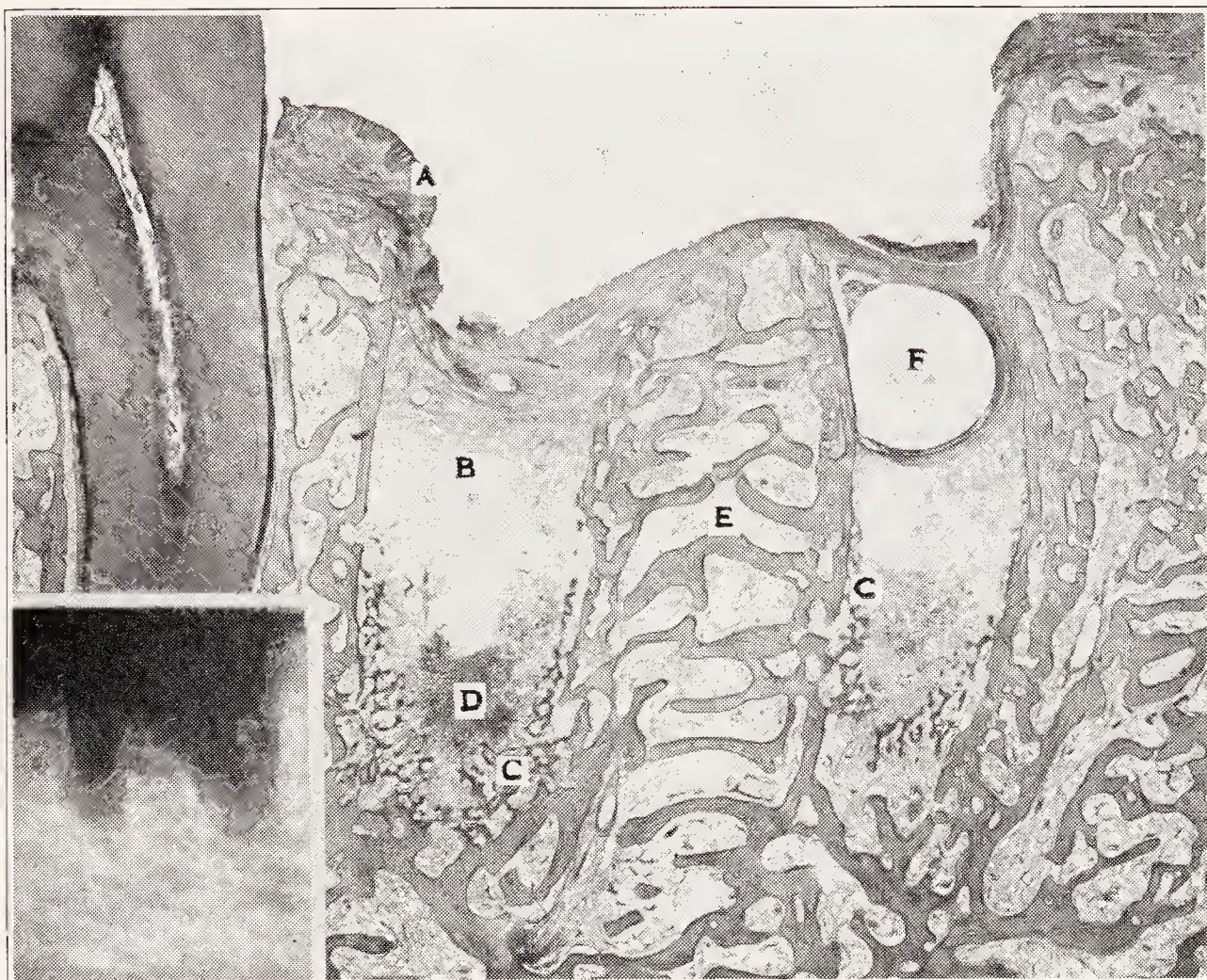


FIG. 409.—Extraction wound, six weeks old, of a lower second molar in man. *A* epithelium; *B*, scar tissue; *C*, new bone in fundus; *D*, remnants of original blood clot; *E*, horizontal spicules of interradicular bone; *F*, abscess cavity. Insert: Radiograph of extraction wound. (Claffin, Jour. Am. Dent. Assn.)

ance. This condition is accompanied by pain, sometimes severe and of neuralgic character; the surrounding gingiva is inflamed, a foul odor may be present, and there may be swelling of the regional lymph nodes. These clinical symptoms usually persist with varying intensity for a week or ten days; then they subside and the wound heals. Frequently small spicules of necrotic bone are eliminated from the wound; on rare occasions the entire bony socket may be exfoliated. The region where dry sockets are most frequent is the posterior portion of the mandible (Krogh).

From the viewpoint of the pathologist a dry socket may be looked upon as a circumscribed osteitis of the alveolar bone with death of bone and formation of small sequestra. The immediate cause for the condition is the invasion of the socket by pathogenic microorganisms. Several circumstances favor the development of such an alveolar bone infection. Difficult extractions³ cause extensive



FIG. 410.—Artificially produced dry socket, ten days old, treated with phenol and collodion. *A*, exposed cribriform plate of socket; *B*, abscess cavity; *C*, granulation tissue; *D*, epithelium; *E*, osteoclasts separating necrotic from living bone. (Claffin, Jour. Am. Dent. Assn.)

tissue injury and are more likely to result in dry sockets than less difficult ones. The blood clot may be disturbed by excessive rinsing or other manipulations, thus depriving the socket of its natural protective covering. Occasionally, however, for no known reason, a dry socket develops after a simple extraction, in spite of all precautions on the part of dentist and patient.

The treatment of dry sockets consists of the application of heat and the use of various drugs to alleviate the pain. None of these drugs, however, noticeably shortens the healing time; apparently it takes a certain length of time—usually about one week—for the body to overcome the bone infection, and little can be done to shorten this time.

Claffin was able to produce dry sockets experimentally in animals by disturbing the blood clot and by introducing bacteria into the socket. Some of the dry sockets were treated with drugs; others were left untreated. The alveolar bone in the sockets became necrotic; extensive inflammatory changes were found in the surrounding marrow spaces. Later, granulation tissue began to proliferate from the alveolar fundus, and osteoclasts separated the necrotic from the living bone (Fig. 410). Finally, the necrotic bone was cast off, the alveolus was filled with granulation tissue, and after considerable delay healing proceeded in a manner similar to that of undisturbed extraction wounds.

HISTOLOGY OF EDENTULOUS JAWS.

Upper Jaw.—Clinical Description of Edentulous Maxilla.—The clinical examination of an edentulous maxilla reveals several zones of varying tissue resistance that are important from the standpoint of the retention of artificial dentures. A firm, resistant tissue of rather uniform thickness covers the crest of the alveolar ridge. This tissue extends from the maxillary tuberosity of one side, over the anterior part of the maxilla to the tuberosity of the other side, and is from 4 to 8 mm. wide. To the labial and buccal periphery of the ridge is attached the movable tissue of the vestibule. This tissue has a layer of submucosa of loose texture that permits wide excursions and changes in the shape of the vestibular tissues during the movements of the lips and cheeks.

The tissue covering the anterior part of the hard palate on the lingual side of the edentulous ridge is dense and resistant. Numerous transverse ridges radiating from the median raphé are usually found in this area. They are called palatine rugæ.

In the posterior portion of the hard palate the mucosa is smooth and elastic. The cushion-like submucosa in this area contains the palatine vessels and nerves, adipose tissue, and numerous glands; the presence of the latter accounts for the constant presence of moisture on the surface of the palatine mucosa.

In the median line of the hard palate, a ridge of great resistance

and sometimes almost bony hardness is frequently found; here the lower surface of the hard palate is covered only by a thin lining of soft tissue. This area, the median raphé or palatine suture, has an outline somewhat like the kernel of an almond with the tapered end pointing anteriorly. The antero-posterior length and width of this hard area vary greatly in different individuals; occasionally there is a bony prominence or exostosis in this region, the torus palatinus (see page 476).

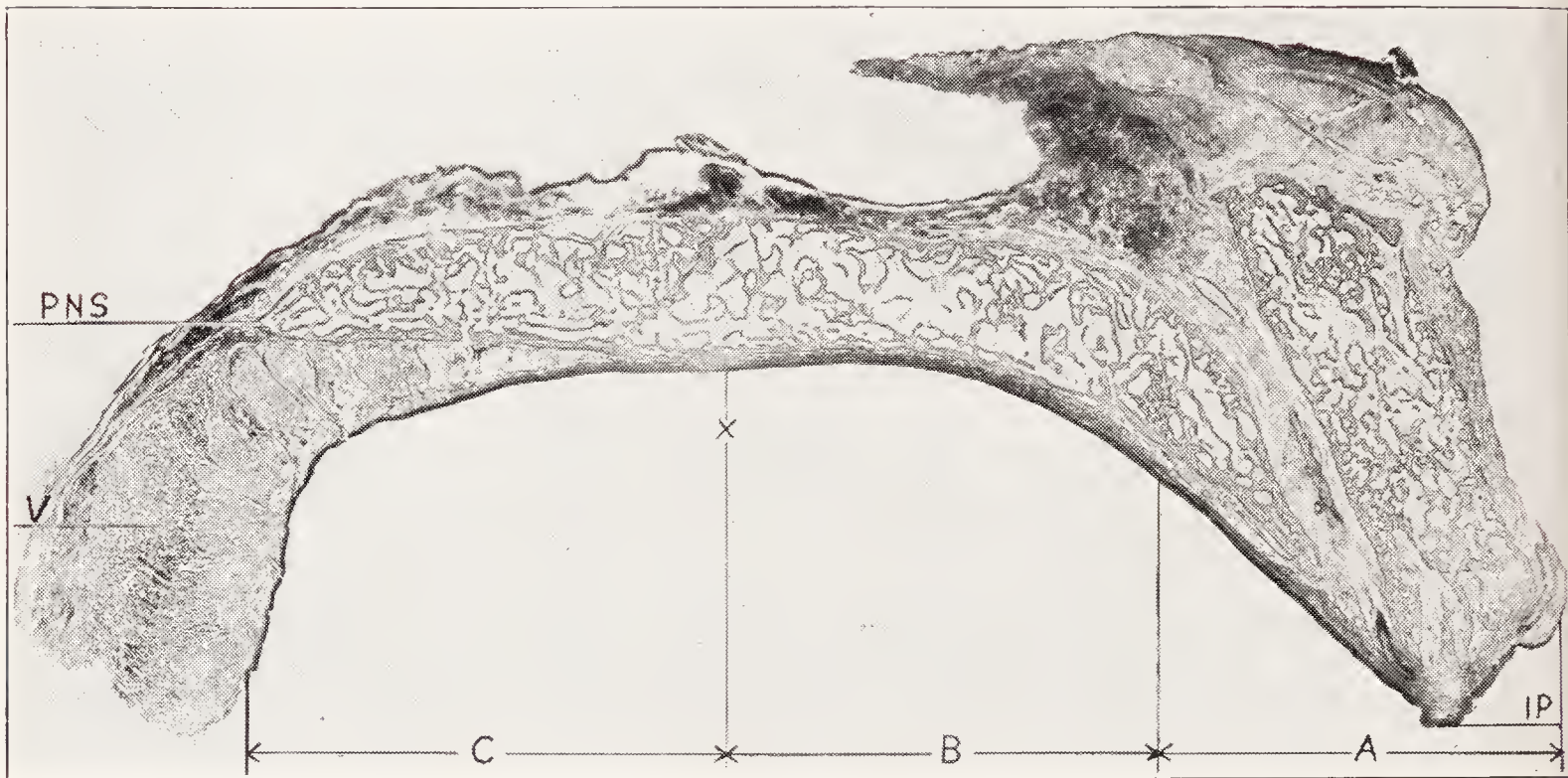


FIG. 411.—Sagittal section through the median line of an edentulous maxilla. A, area of maxillary ridge and incisive foramen; B, hard palate; C, soft palate; IP, incisive papilla; X, ridge of maxillary suture; PNS, posterior nasal spine; V, velum. (Pendleton, Jour. Am. Dent. Assn.)

The posterior border of the denture area is the region where the soft palate or velum is attached to the posterior margin of the hard palate. The line of attachment is indicated by a difference in the appearance and motility of the tissues. The hard palate is grayish in color, while the soft palate is more pink. The soft palate moves in deglutition and phonation; the hard palate is immobile. The line between hard and soft palate curves slightly on both sides from the distal surface of the maxillary tuberosity to the posterior nasal spine, the concavity of the curve pointing backward toward the soft palate.

Histology of Edentulous Maxilla.—The general arrangement of the tissues of the palate in the median line is illustrated by a sagittal section through an edentulous maxilla (Fig. 411). In the anterior part of the specimen a dense layer of fibrous connective tissue covers

the edentulous maxillary ridge. Farther back toward the hard palate, the soft tissue lining of the palate becomes less fibrous, and fat tissue appears in the submucosa. At the height of the vault, the soft tissue covering the lower surface of the palatine bones is very thin, but from there back, it rapidly becomes thicker and drops down, forming the transition to the soft palate. The bone extends



FIG. 412.—Labio-lingual section through an edentulous ridge near the median line in the area formerly occupied by the upper central incisors. *ANS*, anterior nasal spine; *CT*, loose connective tissue on labial side of ridge; *V*, vestibule; *MA*, labial muscle attachment; *MR*, maxillary ridge; *R*, rugæ; *FT*, fat tissue; *IC*, incisive canal; *NS*, nasal septum. (Pendleton, Jour. Am. Dent. Assn.)

backward in a horizontal plane from the height of the vault to the posterior nasal spine.

A higher magnification of the edentulous ridge of another jaw in the median line shows the attachment of the labial muscles to the anterior surface of the alveolar process and the loose movable tissue on this surface (Fig. 412). The ridge itself is covered by fibrous

connective tissue. Farther toward the lingual side fat tissue is present in the submucosa of the hard palate; the irregular outline of the surface is caused by the palatine rugæ. These rugæ are prominences of densely interwoven fibrous connective tissue covered by stratified squamous epithelium.

In a frontal section through the entire edentulous maxilla, in the area of the upper first molar, the general distribution of the soft tissues over the bony skeleton of the palate can be studied (Fig. 413). On both sides, the buccinator muscle is attached to the outer surface of the maxilla. The crest of the bony ridge is covered by fibrous connective tissue. Lingually to the crest the bone recedes; between the lingual surface of the alveolar ridge and the roof of the palate it

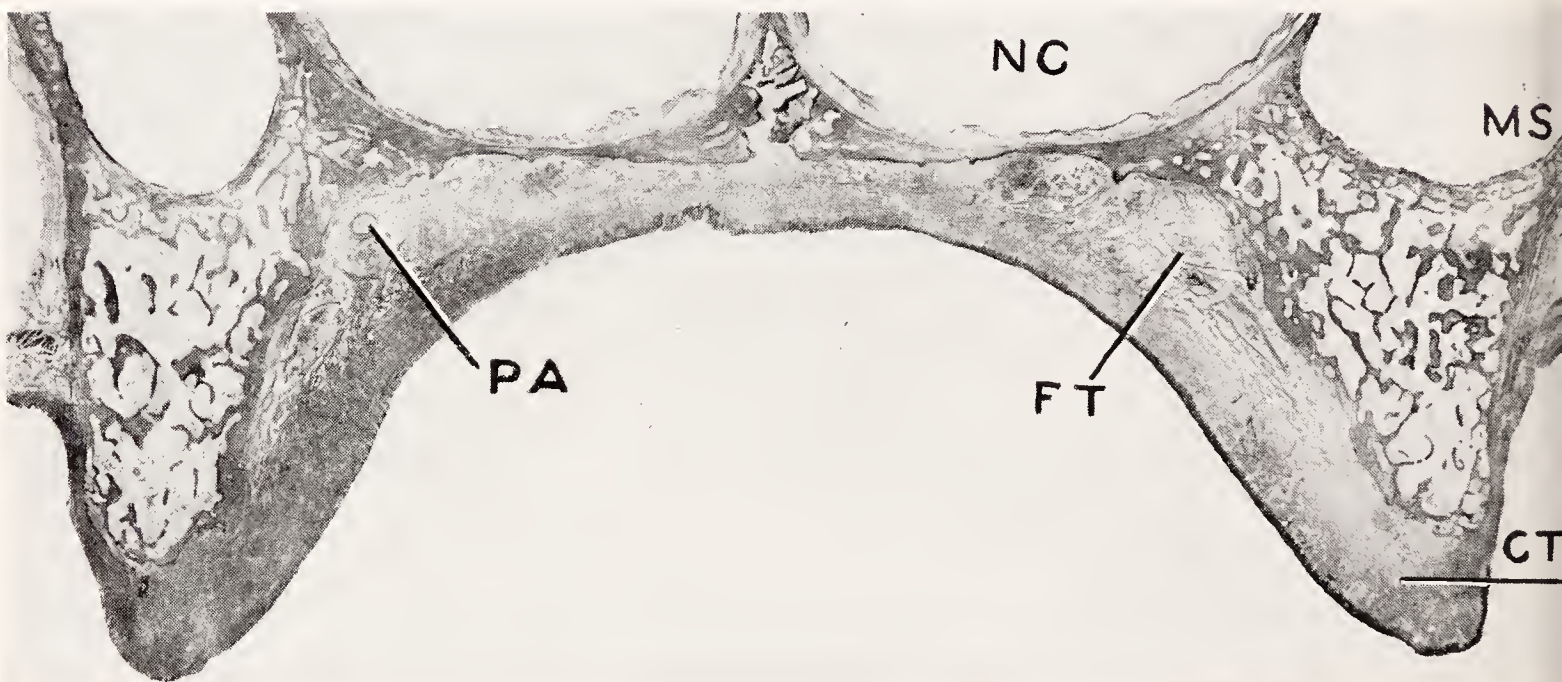


FIG. 413.—Frontal section through an edentulous maxilla in the region of the upper first molar. *NC*, nasal cavity; *MS*, maxillary sinus; *CT*, fibrous connective tissue on crest of ridge; *FT*, fat tissue of palate; *PA*, palatine artery. (Pendleton, Jour. Am. Dent. Assn.)

forms an angle in which the blood-vessels and nerves of the palate are located. The mucosa is stretched from the crest of the ridge toward the vault, forming a triangular space on either side of the median line that is filled with fat tissue and mucous glands.

Posterior to the first molar region the number of glands increases considerably. In a frontal section through the third molar area of the same jaw (Fig. 414), large masses of glands occupy the entire space between the median line and the alveolar ridges; the latter are covered with fibrous connective tissue of considerable thickness. The bone on the buccal side of the ridges protrudes as a distinct prominence. In the vestibule the muscle attachment is identical to that in the preceding illustrations.

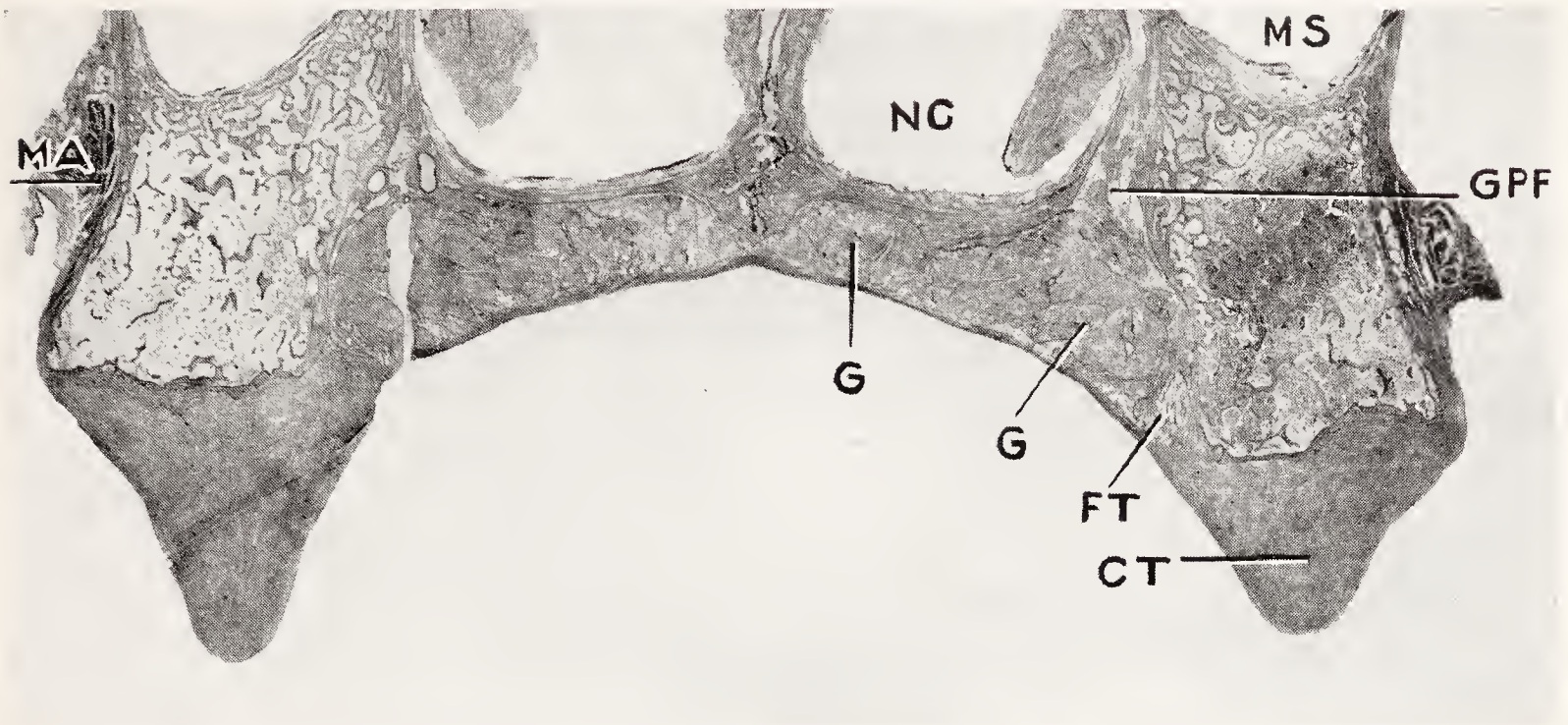


FIG. 414.—Frontal section through an edentulous maxilla in the region of the upper third molar. *NC*, nasal cavity; *MS*, maxillary sinus; *GPF*, greater palatine foramen; *CT*, fibrous connective tissue on crest of ridge; *FT*, fat tissue; *G*, glands of palate; *MA*, buccal muscle attachment (buccinator muscle). (Pendleton, Jour. Am. Dent. Assn.)

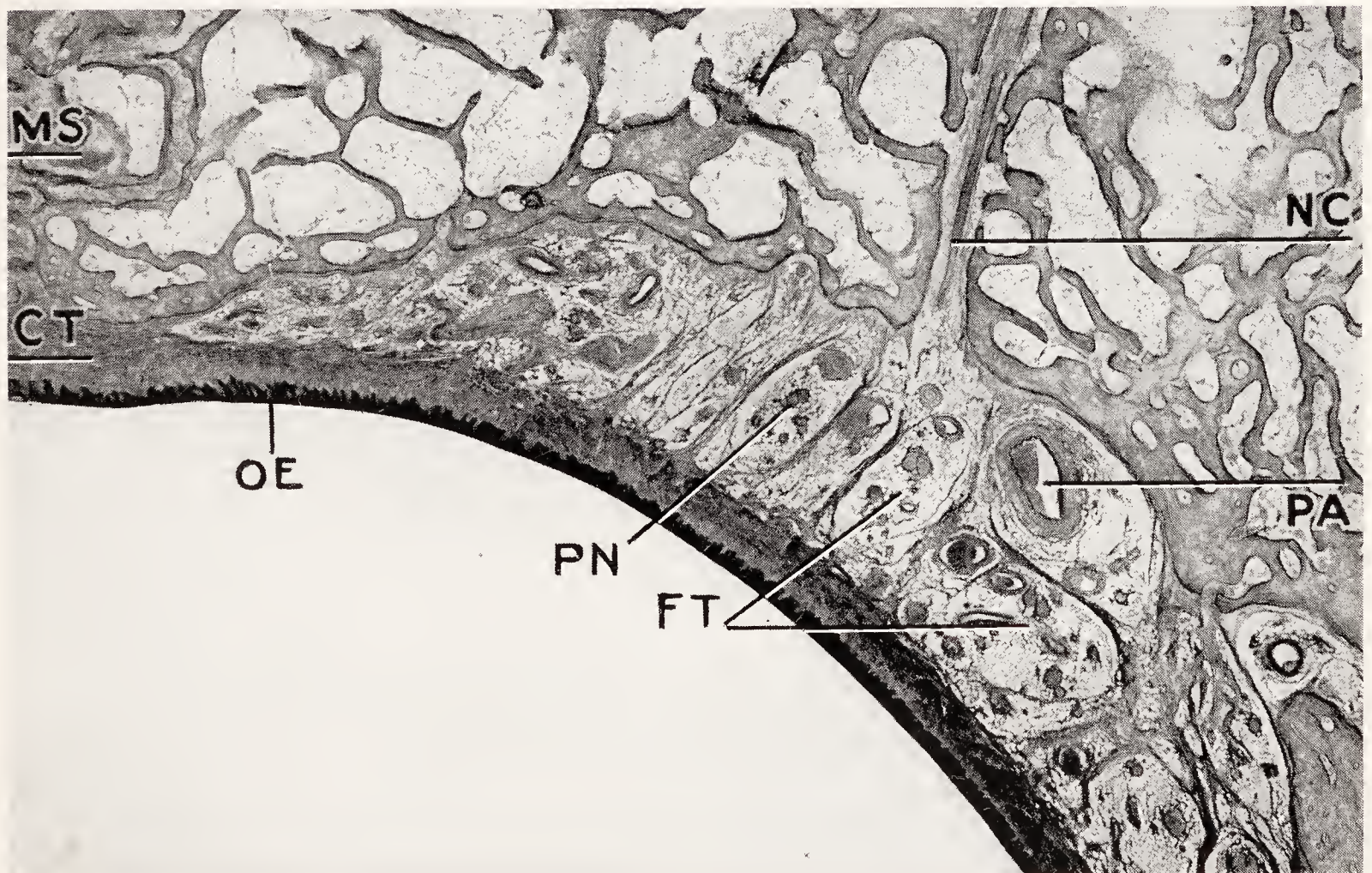


FIG. 415.—Area between maxillary ridge and maxillary suture. *PA*, palatine artery; *PN*, palatine nerves; *FT*, fat tissue; *CT*, fibrous connective tissue overlying the maxillary suture; *MS*, maxillary suture; *NC*, nutritional canal in the bone; *OE*, oral epithelium. (Pendleton, Jour. Am. Dent. Assn.)

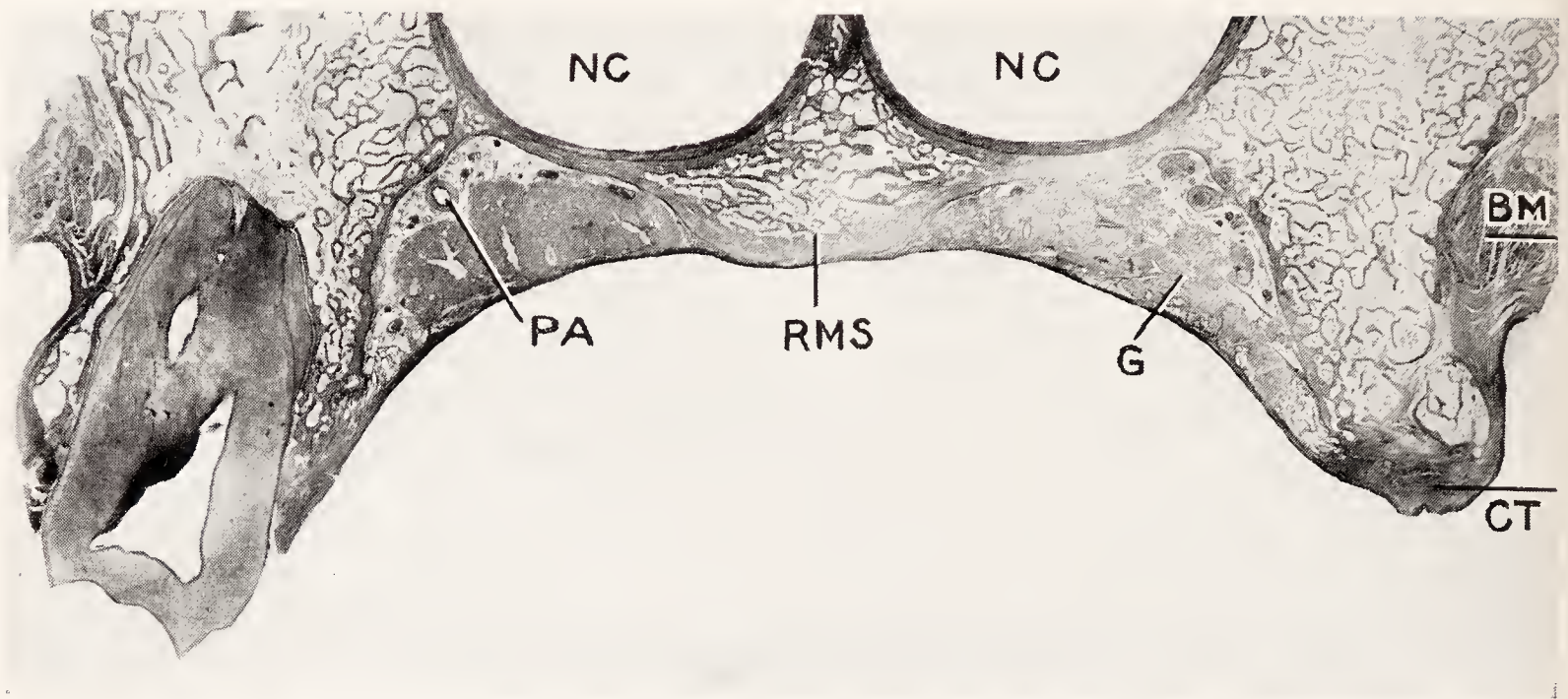


FIG. 416.—Frontal section through an upper jaw in the third molar region. The upper left third molar is intact; the right side of the maxilla is edentulous. *NC*, nasal cavity; *BM*, buccinator muscle; *CT*, fibrous connective tissue; *G*, mucous glands of palate; *RMS*, bony ridge of maxillary suture; *PA*, palatine artery. (Pendleton, Jour. Am. Dent. Assn.)

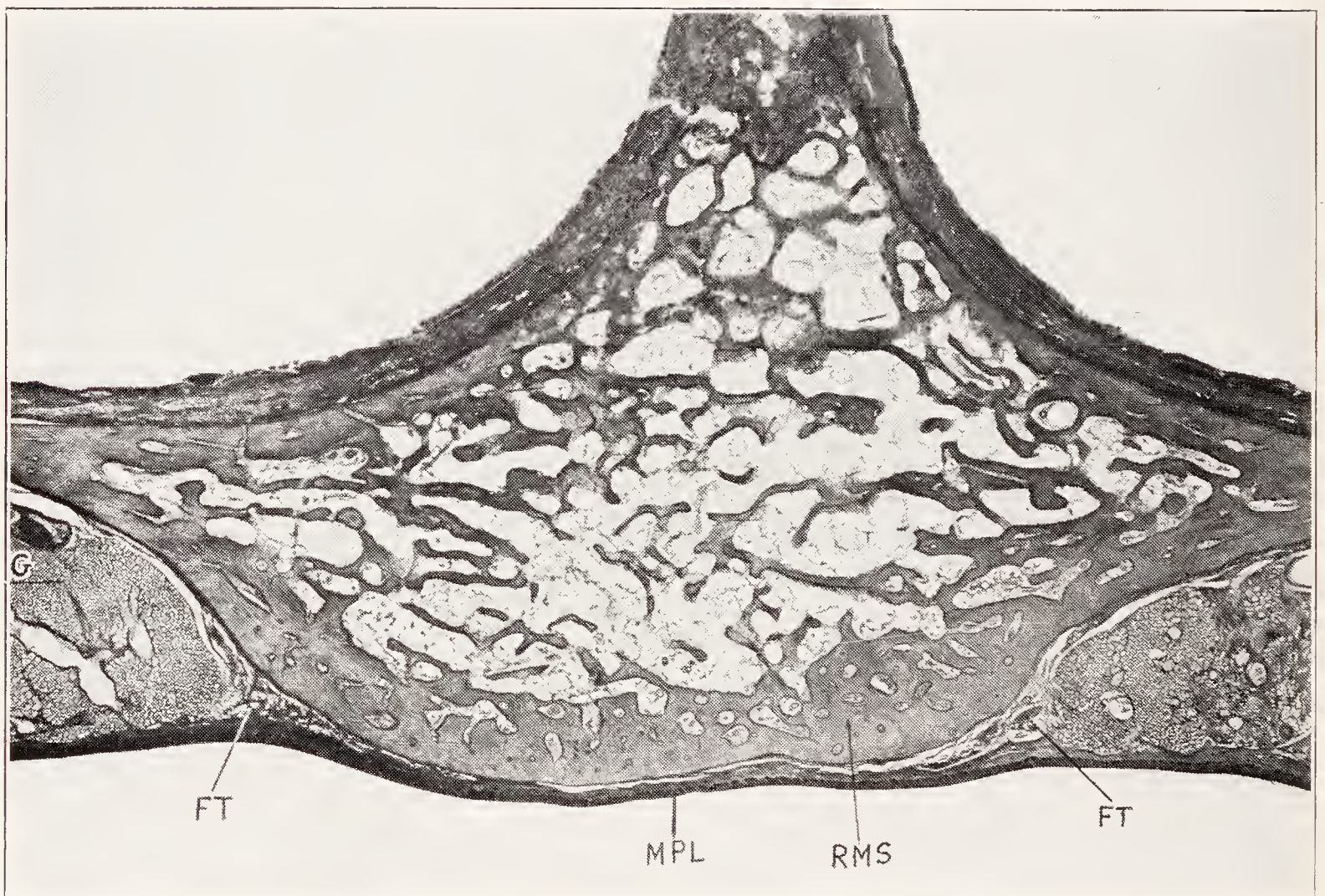


FIG. 417.—Higher magnification of the central portion of Figure 416. Marked bony prominence in the median line of the hard palate. *G*, mucous glands of palate; *FT*, fat tissue; *RMS*, bony ridge of maxillary suture; *MPL*, thin mucoperiosteal lining covering the bone in the median line. (Pendleton, Jour. Am. Dent. Assn.)

In the anterior portion of the upper jaw the triangular area between the residual alveolar ridge, the palatine process of the maxilla, and the soft tissue surface is filled with adipose tissue. Fibrous bands extend upward to the periosteum on the inferior surface of the palatine bones, dividing the fat tissue into several compartments. Embedded in each of these areas, the large and small vessels and nerve bundles of the palate run parallel to the bone surface (Fig. 415). Near the median line the distance between oral mucosa and bone is shorter, and only fibrous tissue is present between epithelium and bone.

A frontal section through the third molar region shows the left third molar still intact; the opposite side is edentulous (Fig. 416). Large masses of glands lie on both sides between ridge and median line and the prominent bony ledge or raphé in the center of the hard palate. The thickness of the soft tissue overlying the palatine artery on both sides is approximately 7 mm.; the soft tissue covering the bone near the median line is only about 0.3 mm. thick (Fig. 417).

Practical Conclusions from Histological Findings in Edentulous Maxilla.—Because of their physical properties, the areas of the edentulous maxilla that have been described in the preceding paragraphs are of importance in impression taking and denture construction. In clinical terminology the edentulous maxilla has been divided into four areas (Fig. 418):

- (a) Primary stress-bearing area.
- (b) Secondary stress-bearing area.
- (c) Valve-producing area.
- (d) Relief area.

(a) The primary stress-bearing area, which includes the entire maxillary ridge (Fig. 418, *PS*), receives the greatest pressure from the denture under masticatory stress. This area is characterized by dense fibrous connective tissue that is firm, yet resilient enough to stand considerable pressure without injury.

(b) The secondary stress-bearing area lies between the maxillary ridge and the median raphé (Fig. 418, *SS*); it includes the palatine rugæ in the anterior portion, and fat tissue and glands in the posterior portion of the maxilla. Because of the vulnerability of the glands this area is of secondary importance in carrying the denture; however, the denture may rest upon it to a certain degree.

(c) The valve-producing area (Fig. 418, *V*) extends all around the maxillary ridge on the periphery of the primary stress-bearing area and along the posterior border of the hard palate. On the labial and buccal sides of the alveolar processes and at the posterior

borders of the hard palates this area is characterized by the presence of loose, movable tissue and muscle attachments. Loose connective tissue is unable to bear stress, but it adapts itself closely to the rounded borders of a denture and thus produces the air-tight seal that is indispensable for good denture retention. If the denture rim extends too high into the vestibule and covers the deepest point of the muscle attachment, it impinges upon the muscles and may be displaced by their contractions.

(*d*) The relief area (Fig. 418, *R*) is the median area of the maxilla over the posterior part of the maxillary suture. The layer of soft tissue covering the bone in this field is often very thin and cannot withstand stress or pressure. Besides, if the denture rested upon

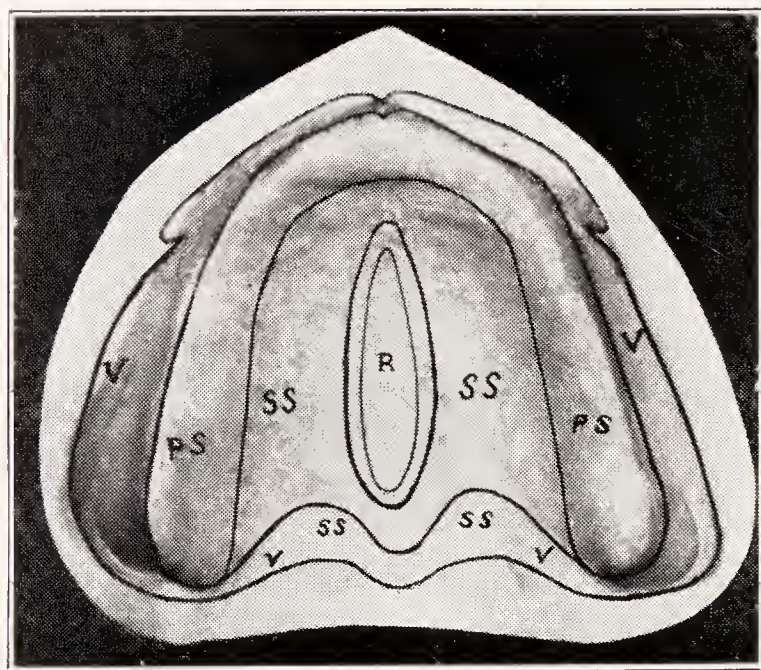


FIG. 418.—Plaster cast of the specimen from which the sections in Figures 412 to 414 were prepared. The areas of denture retention are outlined on the cast. *PS*, primary stress-bearing area; *R*, relief area; *SS*, secondary stress-bearing area; *V*, valve-producing area. (Pendleton, Jour. Am. Dent. Assn.)

the unyielding ridge in the center of the palate, it would rock, since all the surrounding tissues have a much greater compressibility. Therefore, care must be taken to relieve all pressure in the median line by special methods of impression taking.

The amount of fat tissue in the palate is variable; it shows a tendency to increase or decrease as the amount of subcutaneous fat varies elsewhere in the body. It is a common clinical observation that formerly well-fitting dentures become unsatisfactory if the patient loses considerable weight.

Lower Jaw.—The residual ridge of the mandible is covered with dense, fibrous connective tissue that is well suited to support the pressure of a mandibular denture and which, therefore, can be considered the primary stress-bearing area of the lower jaw (Fig. 419, *PS*).

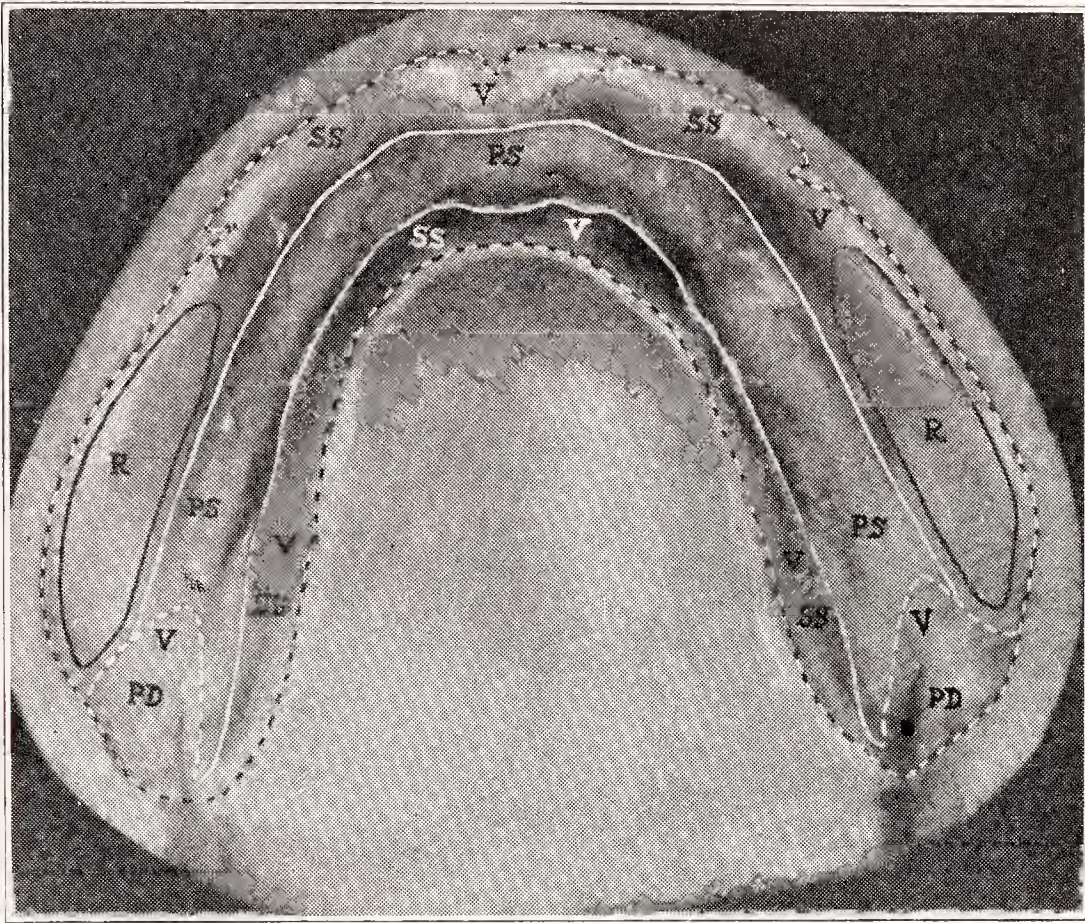


FIG. 419.—Scheme of mandibular area of denture retention. *PS*, primary stress-bearing area; *SS*, secondary stress-bearing area; *V*, valve-producing area; *PD*, post-dam area; *R*, relief area. (Pendleton, Jour. Am. Dent. Assn.)

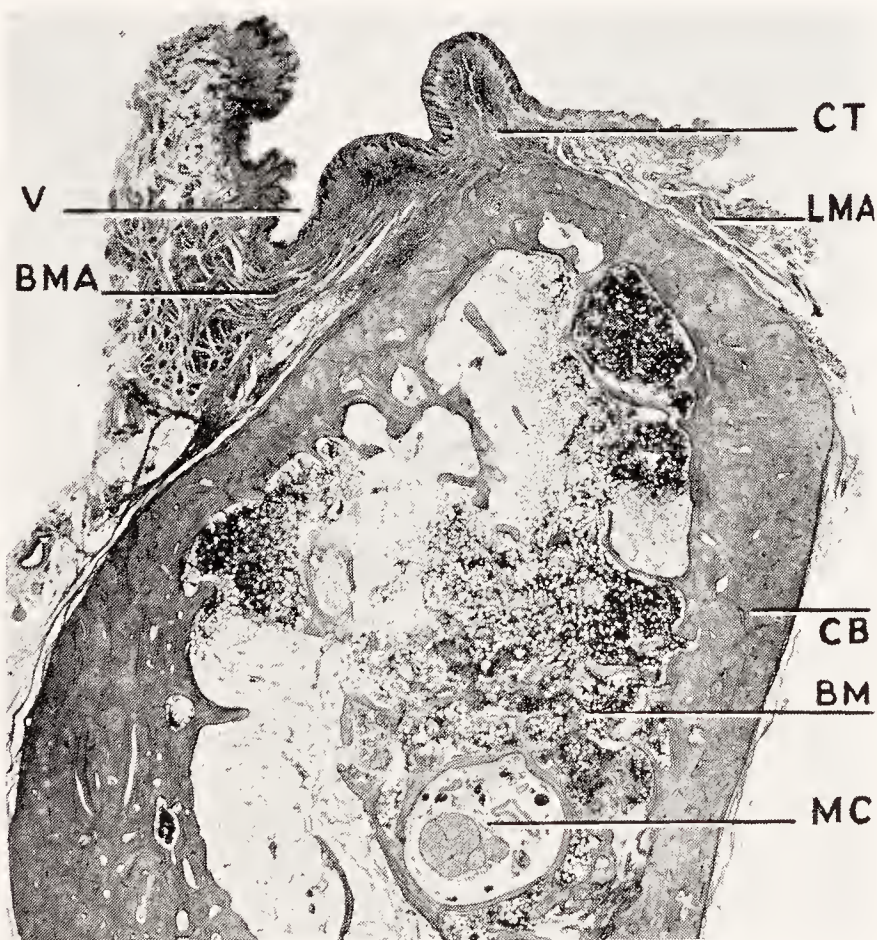


FIG. 420.—Bucco-lingual section through an edentulous mandible in the molar region. *V*, buccal vestibule; *BMA*, buccal muscle attachment; *CT*, connective tissue of residual ridge; *LMA*, lingual muscle attachment; *CB*, compact bone on the surface of the mandible; *BM*, bone marrow; *MC*, mandibular canal containing blood-vessels and nerves. (Courtesy of E. C. Pendleton.)

On the labial and lingual sides of the alveolar ridge are muscle attachments and glands; in these areas there is an abundant amount of loose submucosa that permits extensive positional changes whenever the lips, cheeks, or tongue move. This area of loose tissue can be considered both as the secondary stress-bearing area and as the valve-producing area: it has to carry some of the load of the denture and at the same time provides the peripheral valve seal for the denture (*V* and *SS*, Fig. 419). In the posterior portion of the buccal surface the external oblique ridge forms a bony prominence covered only by a thin layer of mucosa; this tissue arrangement resembles that found in the midline of the palate and like the latter has been designated as an area of relief (Fig. 419, *R*), on the underlying tissues of which the denture must not exert pressure.

Sections through human edentulous mandibles reveal a wide variation in the development of the residual ridge. If this ridge is well formed and fairly high, the denture rests on considerable surface area, supported by fibrous connective tissue (Fig. 420). In jaws with advanced atrophy of the ridge, the attachments of the mylohyoid muscle on the inside and of the buccinator muscle and orbicularis oris on the outside may be located close to each other near the flat surface of the mandible; the denture then rests on movable muscle tissue, a condition that can make it difficult or almost impossible to obtain retention.

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CHAPTER XX.

VINCENT'S INFECTION. DISCOLORATIONS AND STAINS. COMMON DISEASES OF THE ORAL MUCOSA.

VINCENT'S INFECTION.

VINCENT'S infection of the gingivæ is an acute or subacute destructive inflammation of the gingival margin and of the interdental papillæ. The condition is also known as ulcero-membranous gingivitis, necrotic gingivitis, or trench mouth.

The onset of acute Vincent's infection is sudden. It usually begins around one or two teeth and from there spreads to the adjacent gingivæ. At first the gingivæ are acutely inflamed, swollen, and edematous. Soon sloughing of the crests of the interdental papillæ occurs, so that the tissues between the teeth appear punched-out or crater-like. A grayish-yellow pseudo-membrane, consisting of coagulated fibrinous exudate, covers the necrotic surface of the ulcerations. Beneath these ulcerations the gingiva is bright red or bluish-red (Fig. 421). The ulcerated gingival surfaces are painful and very sensitive to touch. Gingival hemorrhages occur readily. There is a characteristic putrid odor from the mouth and a greatly increased flow of saliva. The submaxillary lymph nodes are swollen and tender. The temperature is almost always above normal, in severe cases 103 or 104° F.

Sometimes the sloughing of the gingival crests is not so pronounced; instead merely a fine gray necrotic line runs along the gingival margin. Otherwise the gingivæ are hypertrophic, swollen, and tender, and bleed easily (Fig. 422).

The severity and duration of acute Vincent's infection vary widely. The inflammation may ultimately resolve completely. If the gingival necrosis was extensive, it may leave the cervical portion of the teeth denuded and exposed. In other cases the inflammation may enter a subacute or chronic stage. Then both the objective and the subjective symptoms may be very mild. The condition often resembles a simple non-specific gingivitis. However, in Vincent's infection the interproximal location of the lesions, the presence of truncated papillæ the tips of which have sloughed off, and the absence of suppuration help in making the differential diagnosis. Acute attacks recur frequently in the course of subacute or chronic Vincent's infection.

In smears from mouths of patients with acute or subacute Vincent's infection, two distinct types of microorganisms are invariably

observed in large numbers, a spirochete and a fusiform bacillus (Fig. 423). The spirochete is classified as *Treponema Vincenti* or *Borrelia Vincenti*; it is from 8 to 20 micra long, about 0.4 micra wide, and shows from 3 to 6 loose spirals. The fusiform bacilli are subdivided and classified differently by various investigators. These



FIG. 421



FIG. 422

FIG. 421.—Acute destructive Vincent's infection. When the necrotic interproximal papillæ, especially in the lower jaw, were wiped away, the gingivæ had a punched-out appearance. Beyond the serpiginous, gray necrotic line, the gum is highly inflamed. There is marked hemorrhage, especially on the left side. Blood may be seen oozing from between the lower left lateral incisor and the cuspid.

FIG. 422.—Acute hypertrophic Vincent's infection. In contrast to the destruction seen in Figure 421, there is hypertrophy of the entire gingiva, which was tender and bled easily. A thin gray necrotic line may be seen along the margin of the upper gingivæ. The hypertrophy was of recent onset and disappeared with the other symptoms in response to treatment. (Hirschfeld, Jour. Am. Dent. Assn.)

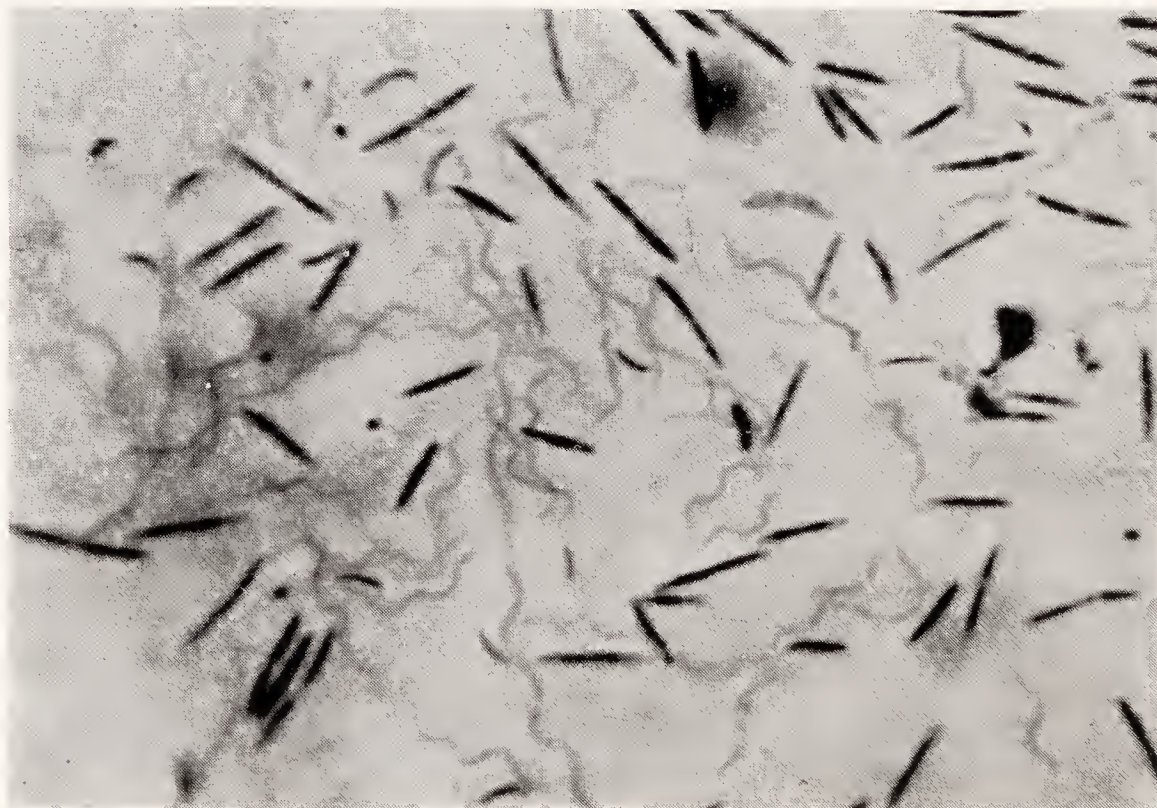


FIG. 423.—Smear from a patient with acute Vincent's infection, showing spirochetes and fusiform bacilli. (Courtesy of E. D. Coolidge.)

bacilli are from 3 to 8 micra long and from 0.3 to 0.8 micra wide, straight or slightly curved, with pointed or rounded ends. In addition, a few cocci and other bacterial forms are usually present in the smears.

The pathogenicity of these microorganisms is doubtful. They are found in apparently healthy mouths, and there is no definite correlation between their form, number, or other characteristics and the clinical manifestations of Vincent's infection. Therefore, mouth smears in Vincent's infection are of little diagnostic value. The clinical symptoms are the only reliable criteria on which diagnosis and treatment can be based.

In view of the foregoing observation it can be understood that underlying or predisposing factors are of decisive importance in the development of the disease. Some investigators go so far as to deny completely the contagious character of Vincent's infection. It is difficult to explain why microorganisms that are present in practically every human mouth should at one time produce severe clinical symptoms and at another time be apparently entirely harmless. It seems that any condition, whether it be traumatic, metabolic, toxic, or infective, may reduce the resistance of the gingival tissues to a point where the ordinarily saprophytic microorganisms become pathogenic and invade and destroy the tissues. Lack of oral care is the main predisposing factor because it favors the development of all kinds of organisms and reduces the local tissue resistance. It is probably true that Vincent's infection cannot be communicated to a person in good health, with a clean mouth and healthy gingivæ.

So-called epidemics of Vincent's infection may be nothing but a simultaneous appearance of the disease among people living under identical dietary and other external conditions. The widespread occurrence of Vincent's infection among the soldiers during the World War ("trench mouth") may well have been due to the fact that all these men lived under the same unfavorable conditions, lack of personal cleanliness, improper or insufficient food, exhaustion, and physical and mental strain, and that thus their resistance was equally lowered. This, however, does not mean that any precautions against spread of the disease should be neglected, such as individual drinking glasses, eating utensils, towels, etc., for patients with acute Vincent's infection.

Several types of treatment have been recommended for this infection. Apparently some patients respond better to one type of treatment than another. General sanitation of the mouth is of paramount importance. Unless all deposits on the teeth are removed in

the course of treatment, a recurrence is almost certain. The drugs used in the treatment of Vincent's infection can generally be divided into the following four main groups: oxidizing agents, dyes, caustics, and spirocheticidal drugs. A detailed discussion of these drugs is contained in the references given at the end of this chapter.

DISCOLORATIONS AND STAINS.

Deviations from the normal color of teeth are classified as intrinsic and extrinsic stains. Intrinsic stains are those that discolor the tooth from within, while extrinsic stains come from without and affect the tooth surface.

Intrinsic Stains.—An internal stain or discoloration may be caused by various substances, such as blood pigment, metals, or drugs. A congenital anomaly characterized by tooth discoloration, hereditary opalescent dentin, has been described in Chapter I.

Blood pigment and the various products of hemoglobin decomposition cause a discoloration that varies from a reddish or yellowish color to slate-gray or bluish-black. The blood pigment enters the dentinal tubules as a result of hemorrhages in the pulp chamber, root canal operation, or necrosis and decomposition of the pulp tissue. Frequently a discoloration is observed following a trauma, with or without tooth fracture. A slight change in color in such a tooth does not necessarily mean that the pulp has become necrotic. Sometimes it is the result of capillary pulp hemorrhages, which cause a temporary discoloration but which are later on absorbed, so that the pulp retains its vitality. After a trauma, if a discoloration becomes more and more pronounced, it indicates that the pulp has died and is decomposing.

The crown of a tooth that has become discolored by blood pigment can sometimes be bleached by strong oxidizing drugs, such as hydrogen peroxide, after the root canal has been thoroughly cleaned and filled.

Silver or copper amalgam fillings may cause a bluish-black or greenish-black discoloration due to the formation of silver sulphide or copper sulphide that gradually stains the dentin and enamel by diffusion. However, with modern amalgams and correct cavity preparation such discoloration rarely occurs.

Among the drugs that may cause a discoloration of the tooth from within are silver nitrate and other silver salts. Indiscriminate use of silver nitrate in root canals or in deep cavities causes a permanent black discoloration of the tooth structures by the precipitation of particles of metallic silver.

Extrinsic Stains.—Stains on the tooth surface may be green, brown, black, or red, or a combination of any of these colors.

Green Stain.—A green discoloration of the enamel is sometimes observed in the deciduous and permanent teeth of children and young adults. The stained area is found mainly on the labial surfaces of the anterior teeth, following the outline of the gingival margin in a crescent-shaped form. The green deposit is firmly attached to the enamel surface and can be removed only with coarse pumice or with a sharp scraper. Often the enamel beneath the green deposits is slightly decalcified.

Lack of oral care is a predisposing factor of this condition. The green color is probably caused by a pigment produced by bacterial growth on the enamel surface and in the superficial, decalcified layers of enamel. A relatively high incidence of green stain is observed in the teeth of children suffering from scrophulosis (tuberculosis of the cervical lymph nodes) and other tuberculous lesions.

Metallic Stains.—Metals and metal salts may cause dark brown, black, bluish, or reddish discoloration of the teeth. Workers in copper, brass, or bronze frequently show a green or bluish-green discoloration of the anterior teeth, caused apparently by continued exposure to metal dust in the air. A brownish color of the teeth has been observed in iron and nickel workers. Mercury and manganese salts, if taken orally over a long period of time for therapeutic purposes, may cause black deposits upon the teeth.

Red Stain.—Red deposits on the teeth are very rare. They occur in poorly cared-for mouths and are due to the presence of chromogenic fungi or bacteria on the tooth surfaces, which produce a red pigment.

Tobacco Stain.—The most common stain on the teeth is that caused by tobacco. Most smokers have a brown or black discoloration on the lingual and sometimes also on the labial surfaces of the anterior teeth. The color is caused by tar products of tobacco combustion and not, as some patients believe, by nicotine, which is a colorless, easily soluble substance.

The dark discoloration of smokers' teeth has no detrimental effect upon the teeth. It can be removed by thorough scaling and polishing with pumice. In older patients who have been smoking all their lives the stain may have actually penetrated into the superficial layers of the enamel; then it cannot be completely removed.

On rare occasions stains similar to those caused by tobacco appear on the teeth of individuals who do not use tobacco in any form. The etiology of this stain is unknown.

COMMON DISEASES OF THE ORAL MUCOSA.*

Herpes Labialis (Cold Sores, Fever Blisters).—Herpes labialis or cold sore is an acute infectious disease, caused by a filtrable virus. It appears suddenly on the lips, beginning usually at the vermilion border. After a short preliminary stage of tension and slight soreness, small vesicles appear on a reddened spot. These vesicles contain a colorless or yellowish fluid. They rupture, and then the lesion begins to dry up and forms yellow adherent crusts. After one or two weeks the cold sore disappears without leaving a scar. Occasionally it occurs on the mucous membrane of the tongue or cheek. The disease is slightly contagious.

Herpes labialis usually accompanies some general illness or disturbance, such as a cold, menstruation, gastro-intestinal disturbances, or emotional strain. In many of the severe febrile diseases, such as pneumonia, herpes labialis is a regular symptom. No specific treatment is known. Mild antiseptics may be applied in the early stages, and ointments may help in removing the crusts during healing.

Aphthæ (Aphthous Stomatitis, Canker Sores).—A simple aphtha is an acute lesion of the oral mucosa. Its etiology is unknown; it is not contagious. It appears suddenly as a single small vesicle, which soon ruptures and leaves a superficial, slightly raised, round or oval erosion that is covered with a fibrinous exudate. The color of the erosion is gray or yellowish; it is surrounded by an area of intense redness of the mucosa. The usual diameter is 2 to 3 mm. The most common locations are the inside of the lips and the base of the tongue.

The outstanding clinical symptom is the severe pain and tenderness that is entirely out of proportion to the size of the lesion. Every movement of the lips or tongue may be very painful; chewing may be almost impossible. There is an increased flow of saliva.

These symptoms disappear gradually in the course of a week or ten days, as the erosion becomes covered with a layer of new epithelium. There is a tendency toward reoccurrence.

Canker sores frequently accompany periods of physical and mental strain or disturbed digestion. In some persons they appear after eating certain foods, which suggests allergy as an etiological factor. The treatment is symptomatic, tending to ease the pain. A phenol or zinc chloride solution, if placed upon the erosion, will relieve the painful symptoms.

* For a complete account of the diseases of the oral mucosa and mouth, see Prinz and Greenbaum, *Diseases of the Mouth and Their Treatment*, Philadelphia, Lea & Febiger, 1939, and Cornbleet, *Selected Diseases of the Mouth*—in Gordon, *Dental Science and Dental Art*, Philadelphia, Lea & Febiger, 1938.

Thrush.—Thrush is an inflammation of the oral mucous membrane caused by an infection with a yeast-like fungus, *Monilia albicans*. It occurs almost exclusively in young infants, especially in sick and poorly developed infants with low resistance. The lesions are whitish, smooth, sharply outlined, slightly raised patches on the inflamed mucosa of the tongue, cheeks, or palate. This growth can easily be scraped off, which leaves behind a raw, bleeding surface. If the scrapings are examined microscopically they are found to consist almost wholly of a dense felt-like mass of threads of the thrush fungus.

Thrush is a contagious disease. Before the era of general sanitation and milk hygiene, thrush epidemics were a serious problem among the infants in orphanages and foundling homes. The treatment consists of careful cleaning of the mouth, the application of mild antiseptics, and improvement of the general health and resistance of the patient.



FIG. 424.—Leukoplakia of the tongue.
(Waldron in Bunting.)

Leukoplakia.—Leukoplakia is a low-grade, chronic inflammation of the oral mucosa with a varying degree of keratinization of the epithelial surface. The lesions in leukoplakia consist of white or bluish-white patches on the mucosa of the lips, cheek, tongue, palate, or alveolar process. The white color is caused by hornification and opacity of the normally transparent, pink mucous membrane. The lesions develop slowly and painlessly over a period of several years. In the early stages the mucosa looks as if milk had been spilled over it; there is no perceptible induration or change in texture (Fig. 424). Later the patches turn dead white, thick, and stiff, and are slightly raised above the surrounding normal mucosa. Eventually they may become rough and fissured, and then they have a tendency to turn into malignancies. From 20 to 30 per cent of all oral epitheliomas develop from pre-existing leukoplakia patches (see p. 472).

The microscopic examination of leukoplakia patches reveals that the epithelium is thickened by an increase in the number of cell rows in the Malpighian layer. The surface is intensely hornified, and the stratum corneum may be piled up (hyperkeratosis). The subepithelial connective tissue shows a varying degree of inflammatory round cell infiltration. The different stages leading from the

normal mucosa via leukoplakia formation to carcinomatous proliferation of the epithelium are shown in Figure 425.

Leukoplakia is primarily a disease of the male sex; its highest incidence is observed during the fifth and sixth decades of life. Chronic irritation is a definite etiological factor. Excessive use of tobacco, in any form, is one of the main causes of it. If the patient chews tobacco regularly, a leukoplakia patch may develop on the

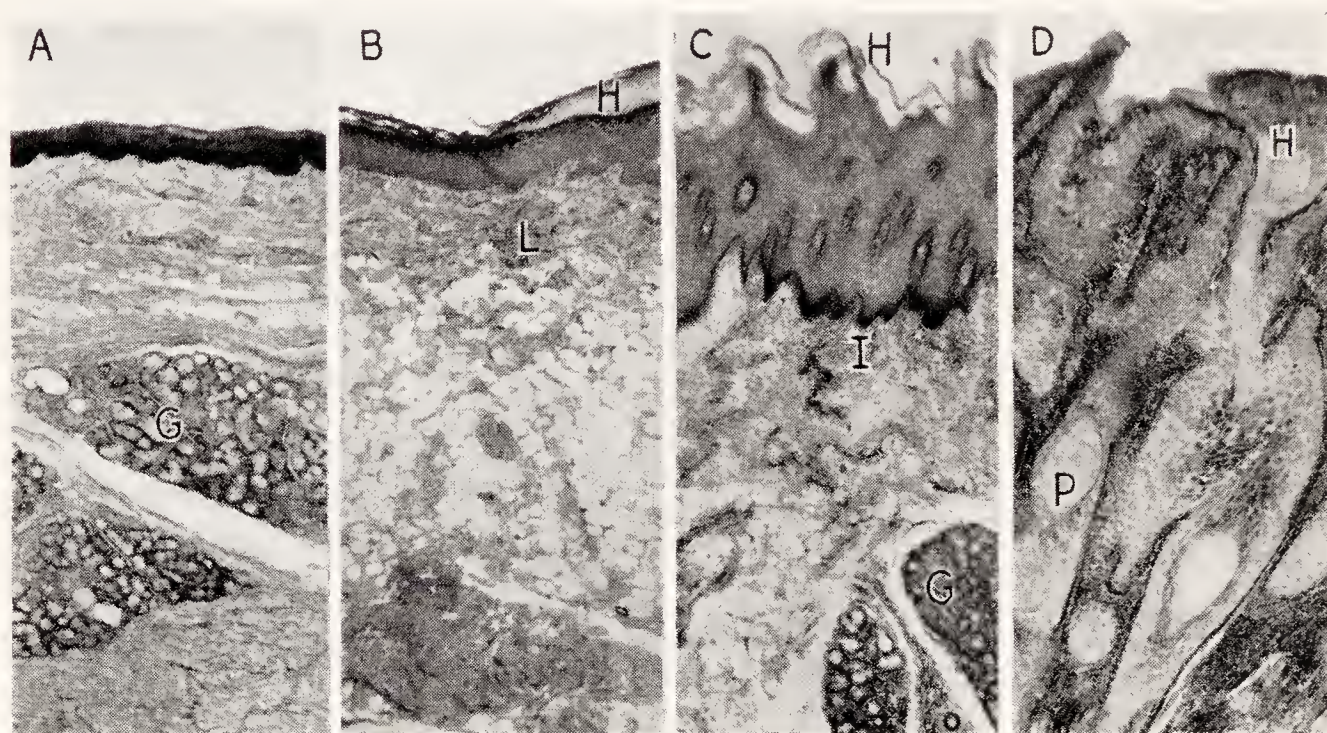


FIG. 425.—Progression from normal mucous membrane to carcinoma. The four areas A, B, C, and D illustrate approximately corresponding areas on the inside of the lower lip of middle-aged men. The magnification, $\times 25$, is identical in all four photographs.

A.—Normal mucosa: thin stratum corneum and even border between epithelium and corium. G, glands.

B.—Early leukoplakia: thickened Malpighian layer and stratum corneum (H); irregularity of the under surface of the epithelium; L, slight degree of subepithelial round cell infiltration.

C.—Advanced leukoplakia: greatly thickened Malpighian layer; casting-off of hornified surface cells (H); irregular border between epithelium and corium with long rete pegs growing into the corium. I, round cell infiltration; G, glands.

D.—Squamous cell carcinoma (epithelioma) developing in an area of leukoplakia: irregular surface hornification, H; extensive irregular ingrowth of epithelial columns into the underlying connective tissue with formation of epithelial pearls (P). Intensive round cell infiltration of the connective tissue.

mucous membrane of the area where the tobacco is usually held. In heavy pipe-smokers leukoplakia may occur in that area on the dorsum of the tongue where the pipe stem usually rests. In other smokers white streaks develop on the inside of both cheeks, corresponding to the area that lies between the upper and lower teeth and is exposed to the smoke. The tarry and oily products of tobacco combustion and the sharp juice of chewing tobacco are the irritating agents. Another possible etiological factor is alcohol in excess, and

very hot or highly seasoned foods. Lack of oral care, dirty, broken-down teeth with ragged edges, and poorly fitting dental restorations are known to produce leukoplakia because of the constant mechanical and bacterial irritation of the mucous membrane. Finally, tertiary syphilis must be cited as a predisposing factor, particularly in leukoplakia of the tongue. The exact manner in which syphilis influences the oral mucosa and favors the development of leukoplakia is unknown.

There is no specific treatment for leukoplakia. In the early stages the disease may be completely arrested by elimination of the etiological irritation. Once typical white patches have developed nothing can be done to change them. If they are small and sharply outlined they can be excised surgically. The use of tobacco should always be discontinued. The teeth must be carefully examined, all badly diseased ones extracted, and the remaining ones treated if necessary. Anti-syphilitic treatment should be instituted, if needed. The application of caustics to the leukoplakia patches is decidedly contraindicated; the added irritation is harmful and may even stimulate the development of a carcinoma.

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CHAPTER XXI.

TUMORS OF THE ORAL CAVITY.

THE classification and clinical characteristics of tumors are usually taught in the course of General Pathology. Therefore, in this chapter only a review of the fundamental facts will be given, with special emphasis on the types of tumors occurring in the oral cavity, since this knowledge is important to the dentist.

DEFINITION AND GENERAL CLASSIFICATION OF TUMORS.

A tumor is an abnormal proliferation of tissue cells that grow unrestrainedly and have no useful function. In this respect a tumor is different from the cell growth in repair or inflammation in which the proliferative process serves a definite purpose, namely, that of regenerating lost tissue or combating injury and infection.

Tumors are divided into two classes: benign or innocent, and malignant. The distinguishing characteristics between the two will be put down in the form of a table. However, these distinctions do not hold true under all circumstances; there may be exceptions to any one of them.

TABLE—CLINICAL AND PATHOLOGICAL DIFFERENCES BETWEEN BENIGN AND MALIGNANT TUMORS.

Benign.	Malignant.
Grows slowly	Grows rapidly
Confined or incapsulated; does not infiltrate surrounding tissues	Not confined; invades and destroys surrounding tissues
Does not cause metastasis	Causes metastasis in lymph nodes and in distant parts of the body
Does not recur after removal	Has tendency to recur after surgical removal
Cells closely resemble normal cells or structures	Cells deviate from normal structure and arrangement (anaplasia)
Does not cause cachexia and does not, as a rule, endanger life.	Usually causes cachexia and destroys life, unless properly treated.

CLASSIFICATION OF TUMORS OF THE ORAL CAVITY.

Tumors of Epithelial Origin

Benign: Papilloma (wart)

Adenoma

Malignant: Carcinoma

Squamous-cell carcinoma (Epithelioma)

Basal-cell carcinoma

Adenocarcinoma

Tumors of Connective Tissue Origin

Benign: Fibroma
 Epulis
 Osteoma
 Hemangioma
 Myoma
 Lipoma
 Mixed tumor of parotid

Malignant: Sarcoma
 Spindle cell sarcoma
 Fibrosarcoma
 Osteosarcoma
 Chondrosarcoma
 Lymphosarcoma
 Malignant melanoma

Tumors of Dental Origin

Odontoma
 Ameloblastoma (Adamantinoma)

Tumors and Dermoid Cysts Containing Teeth

This classification does not include all tumors that are observed in the oral cavity, since on rare occasions almost any kind of tumor can occur there. However, these exceptional tumors have little practical significance; and, therefore, only the more common types have been included in the above list.

BENIGN EPITHELIAL TUMORS.

Papilloma.—The papilloma is a small, pedunculated epithelial growth situated on the surface of the skin or the oral mucosa. It is divided into lobes or divisions that give it a lobulated appearance, resembling a very small head of cauliflower. Histologically, it consists of a fibrous core covered with proliferated squamous epithelium (Fig. 426). Frequently the surface is markedly hornified. Such hornified papillomas of the skin or lip are often referred to as warts; however, not every wart is a papilloma; there are other types of warts that are infectious.

Adenoma.—An adenoma is a benign tumor derived from glandular epithelium and closely resembling the structure of a gland. It occurs in the mouth, but is very rare.

Precancerous Lesions.—The term “precancerous lesion” is applied to a group of conditions that are known occasionally to develop into carcinoma. Thus, a precancerous lesion may be defined as a pathological condition which in itself is not malignant

but which may develop into a malignancy. In the oral cavity the following precancerous lesions are observed:

Papilloma.—Sometimes a papilloma becomes a carcinoma. It begins to grow rapidly, invades the underlying tissue, and shows other characteristics of malignancy.

Leukoplakia (see page 467).—Leukoplakia is an important predisposing factor to malignancy. Patches of leukoplakia should always be kept under observation; rapid thickening and piling up of tissue



FIG. 426.—Squamous papilloma growing from skin. (Boyd.)

and indications of inflammation are symptoms that are highly suggestive of the beginning of a change toward malignancy. Statistics indicate that about 25 or 30 per cent of leukoplakias eventually develop into carcinomas.

Cracks and Fissures in the Mucosa.—Deep fissures in the surface of the lip or tongue may give rise to a malignant growth. This is particularly true of the deep cracks in the lower lips of older men, which often persist for years and finally develop into epitheliomas. Chronic glossitis with proliferation and fissuring of the tongue epithelium often precedes carcinoma of the tongue; this condition is sometimes caused by syphilis.

Chronic Irritation and Ulceration.—Chronic ulcers of the oral mucosa may be caused by ragged edges of broken-down teeth, sharp edges of fillings, or by the borders of ill-fitting dentures. Another cause of chronic irritation and ulceration of the mucous membrane is burns caused by smoking. Any of these areas of chronic irritation may later be the site of a malignant growth.

MALIGNANT EPITHELIAL TUMORS.

Epithelioma.—The epithelioma or squamous-cell carcinoma is the most common malignant tumor found in the mouth. It is essentially a disease of the male sex and of advanced age, although

exceptions are known. The favorite site for an epithelioma is the lower lip, then, in the order of frequency, tongue, cheek, jaw, palate, and floor of the mouth. It usually begins as a nodule or slight thickening of the mucosa or skin, with the formation of a scab on the surface. Later the tissue breaks down, and an ulcer with indurated borders forms, which spreads gradually and destroys the surrounding tissue. Metastasis occurs by way of the lymph stream; the submental and submaxillary lymph nodes are the ones primarily affected.

In the early stages, an epithelioma of the mouth or lips can easily be cured by excision, irradiation, or by a combination of various

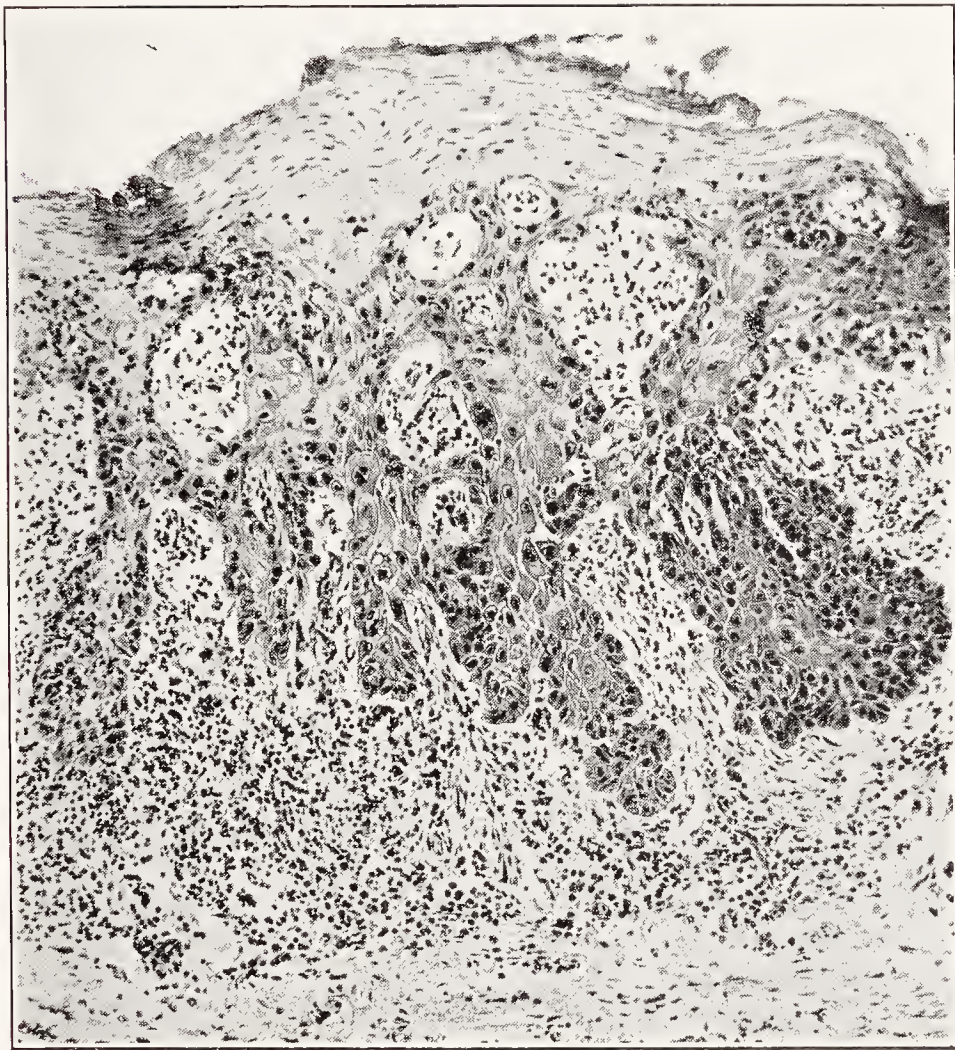


FIG. 427.—Squamous-cell carcinoma of the lower lip, early stage. Note epithelial down-growths and zone of lymphocytes. (Bell.)

methods. The results are very encouraging, and a great number of permanent cures have been recorded. Therefore, the importance of early diagnosis and early and proper therapy cannot be strongly enough emphasized. On the other hand, in the advanced stages cure is uncertain, and the patient may die from cachexia or from complications, such as pneumonia, septicemia, or hemorrhage.

Histologically, an epithelioma shows irregular strands of squamous epithelial cells proliferating into the underlying connective tissue. The latter shows a varying degree of inflammatory round-cell infiltration. Horn pearls are frequently found in the epithelium (Fig. 427).

Basal-cell Carcinoma.—The basal-cell carcinoma or rodent ulcer occurs on the skin of the face of old people. It grows slowly, is of low malignancy, and responds readily to treatment by irradiation. Histologically, the rodent ulcer consists of solid masses of epithelial cells of basal-cell character, proliferating into the dermis. There is no hornification.

Adenocarcinoma.—The adenocarcinoma or carcinoma of glandular origin is rare in the oral cavity. Far more frequently it occurs in other parts of the intestinal tract, in the stomach or the intestines. It is built of cylindrical cells the arrangement of which resembles the structure of glands.

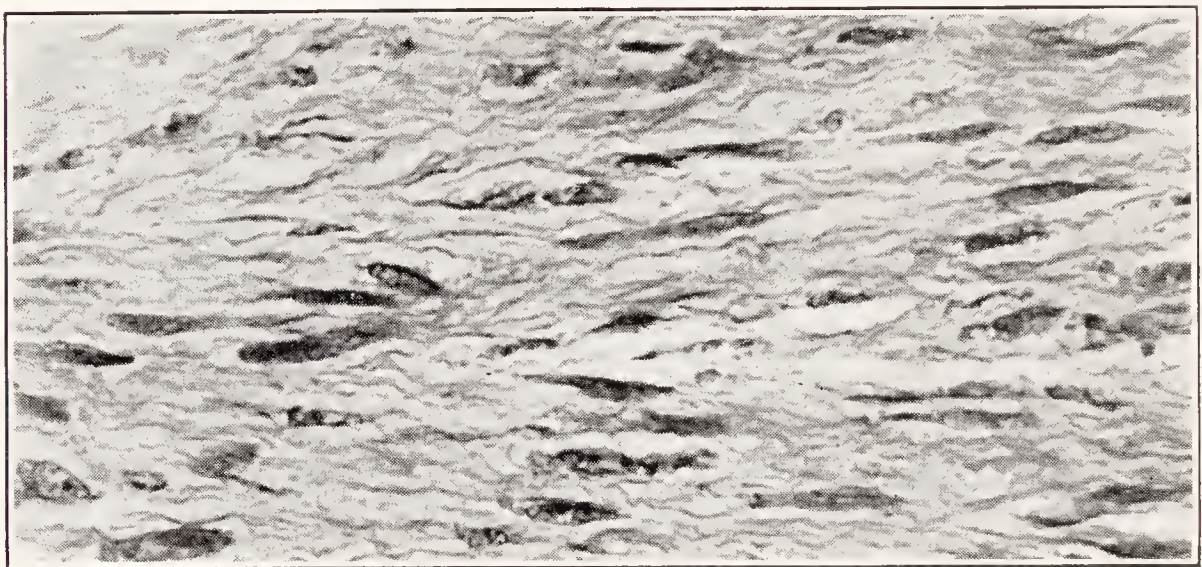


FIG. 428.—Fibroma showing fibroblasts and fibers. (Boyd.)

BENIGN CONNECTIVE TISSUE TUMORS.

Fibroma.—A fibroma consists of fibrous connective tissue covered by a smooth layer of epithelium. It is by far the most common oral tumor and varies in size from several millimeters to 1 or 2 cm. It may be pedunculated or sessile (attached by a broad base). If it contains mostly dense fibrous connective tissue, it is clinically known as a hard fibroma; if it contains more loose connective tissue and vessels, it is called a soft fibroma.

In the oral cavity the most common site for fibromas is the inner surface of the cheeks, the palate, and the alveolar process. There they usually appear as soft, round small tumors covered by normal mucous membrane.

Histologically, a fibroma consists of fibroblasts and bundles of collagenous fibers with a varying number of blood-vessels (Fig. 428).

Epulis.—An epulis is a connective tissue tumor originating from the periosteum of the alveolar bone. Its stroma contains numerous multinucleated giant cells. The typical epulis appears as a firm,

bluish or brownish tumor attached to the alveolar process by a peduncle or by a broad base (Fig. 429). Adjacent teeth are often forced apart or loosened by the tumor. The radiograph reveals destruction of the underlying jaw bone and sometimes areas of calcification within the epulis itself.

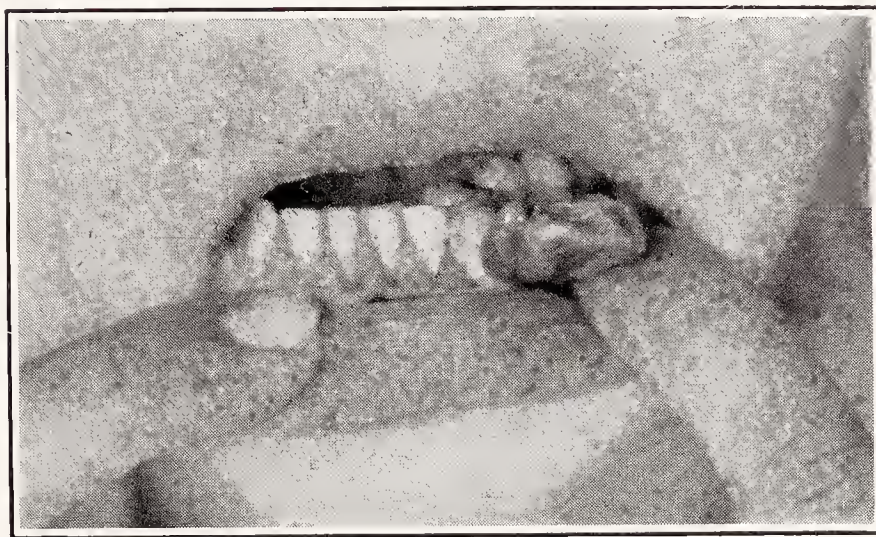


FIG. 429.—Epulis. (Bell.)

The histological appearance of an epulis is that of a cellular and vascular stroma with numerous giant cells, each containing cytoplasm and many nuclei (Fig. 430). Bone is frequently found in the center of the epulis; it shows both resorption (osteoclasts) and formation of new bone (osteoblasts). The surface of the tumor is covered with normal mucous membrane.

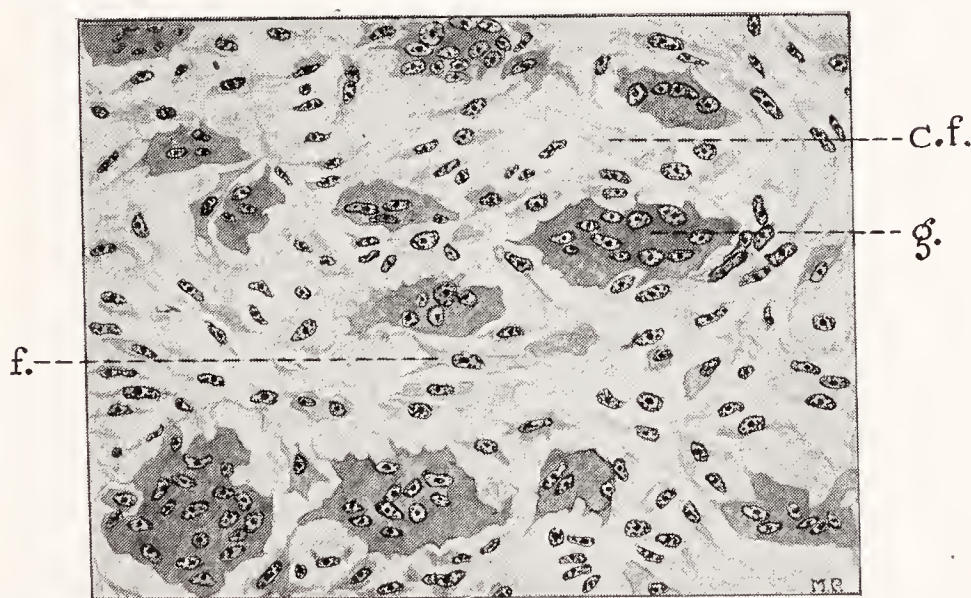


FIG. 430.—Epulis (drawing). Note giant cells, *g*; small fibroblasts, *f*; and collagenous fibers, *c.f.* (Bell.)

Because of the close relationship of the epulis to the underlying alveolar periosteum and bone, the latter must be included in the removal of the tumor to prevent recurrence.

Osteoma.—An osteoma is a benign tumor of the bone tissue. True osteoma is rare; it can greatly deform the maxilla or mandible.

Much more common is an excessive formation of bone, known as *exostosis*. Exostoses frequently occur on the alveolar process; they are painless, hard, bony prominences covered by normal, thin mucous membrane. They may cause considerable annoyance if artificial dentures are constructed over them, and, therefore, under such circumstances they should be removed surgically. A special form of exostosis is an oblong, solid bony ridge occurring in the mid-line of the hard palate, which is known as *torus palatinus*; it may be 2 to 3 cm. long, 1 to 1½ cm. wide, and protrude several millimeters beyond the normal level of the palatine vault.

Hemangioma, Myoma, Lipoma.—Hemangiomas (vascular tumors), myomas (muscle tissue tumors), and lipomas (fatty tumors) have been observed in the mouth, but are rare. A lipoma may appear as a pedunculated appendage to the lips. Hemangiomas occur on the tongue or lips; they are bluish, soft tumors of peculiar sponge-like structure and consist of a conglomeration of enlarged blood-vessels. By digital compression the blood can be expressed from the tumor; when the pressure is released the blood rushes back into the enlarged vessels of the tumor.

Mixed Tumors.—In the salivary glands, especially the parotid, occur tumors composed of various tissues, called mixed tumors. They have also been found in the palate and oral mucous membrane. Mixed tumors may reach considerable size and cause marked deformity of the face.

Such tumors of the salivary glands consist of a mucoid connective tissue stroma in which are embedded strands of epithelial cells in gland-like arrangement, cartilage, and lymphoid tissue. The true nature of these tumors and their origin, whether mesodermal or ectodermal, are uncertain.

MALIGNANT CONNECTIVE TISSUE TUMORS.

Malignant tumors of connective tissue origin are called sarcomas. They are divided into several types according to the histological appearance of the cells of which they are composed. Sarcomas are fast-growing, destructive tumors, generally of high malignancy; they are more likely to develop in young people than are carcinomas, which, as a rule, are found in the middle- and old-aged. Metastasis in sarcoma usually takes place through the blood stream.

Spindle-cell Sarcoma, Fibrosarcoma.—In a fibrosarcoma the fundamental cell is the spindle-shaped fibroblast. It is distinguished from the fibroma, which is also a fibroblastic tumor, by its rapid

and destructive growth and by the formation of metastases. Microscopically the cells of a fibrosarcoma show numerous mitotic figures, indicative of rapid cell proliferation, and an irregular and atypical arrangement of the cells (anaplasia). Fibrosarcoma of the jaw is usually fatal; fortunately it is relatively rare.

Osteosarcoma, Chondrosarcoma.—The osteosarcoma and chondrosarcoma are malignant proliferations of the cells of bone, periosteum, and cartilage. Osteosarcoma of the jaw may reach huge dimensions. The tumor cells retain the power to lay down bone, although the structure is irregular and imperfect; often the bone is uncalcified or preformed as cartilage. A radiograph of an osteosarcoma of the jaw often shows large amounts of newly formed bone radiating from the original jaw bone into the tumor.

Lymphosarcoma.—A lymphosarcoma consists of a reticulum containing small round cells that resemble lymphocytes and other cells related to the lymphatic system. It has been observed in the tongue on rare occasions.

Malignant Melanoma (Melanosarcoma).—The malignant melanoma is a tumor derived from pigment-producing tissue. Clinically, it is characterized by its dark color, produced by the pigment in the cells. Histologically, it consists of cells laden with granules of pigment. Metastases occur frequently and extensively. In the oral cavity, melanosarcoma occurs in the palate, although it is an uncommon tumor in this area.

Other forms of sarcoma have been observed in the oral cavity but so rarely that they are not included in this general review.

TUMORS OF DENTAL ORIGIN.

The two tumors that occur only in the oral cavity and are derived from dental tissues are the odontoma and the ameloblastoma (adamantinoma). Both are true, benign tumors; they are derived from dental tissues in different stages of differentiation and, as a result, vary widely in their clinical and pathological appearance.

Odontoma.—An odontoma is a hard tumor composed of enamel, dentin, cementum, and connective tissue. It may be located entirely inside the jaw, usually in the molar region, or it may protrude from the jaw into the oral cavity. Odontomas are not united with the jaw bone but are separated from it by a capsule of fibrous tissue.

Odontomas grow slowly and painlessly and are often not discovered until they cause distention of the jaw or appear on the

surface. A radiograph reveals a densely calcified mass located in the jaw but separated from it by a clearly outlined soft tissue space. Sometimes the radiograph reveals one or more teeth fused to the odontoma; the latter may be attached to the root of an erupted tooth (radicular odontoma) (Fig. 431) or to the crown of an unerupted tooth (coronal odontoma) (Figs. 432 and 433).

Histologically, odontomas are a more or less irregular conglomeration of all dental tissues. Sometimes they contain multiple small



FIG. 431.—Radicular odontoma attached to lower third molar. The tumor is composed mostly of regular and irregular dentin; only small islands of not yet calcified enamel and some spaces of the dissolved enamel can be seen. (Blum, *Dent. Cosmos*.)

tooth-like structures, each consisting of an outer layer of enamel, a core of dentin, and a central area of connective tissue resembling pulp tissue; these individual, component, small teeth are fused by a cementum-like tissue or by irregular dentin. If such structures are located on the surface of the odontoma, the tumor has a granulated surface, with many small, rounded enamel protuberances.

This type is usually described as a composite odontoma (Figs. 434 and 435). Sometimes the tumor contains larger areas of enamel and dentin, giving somewhat the appearance of one or several dis-

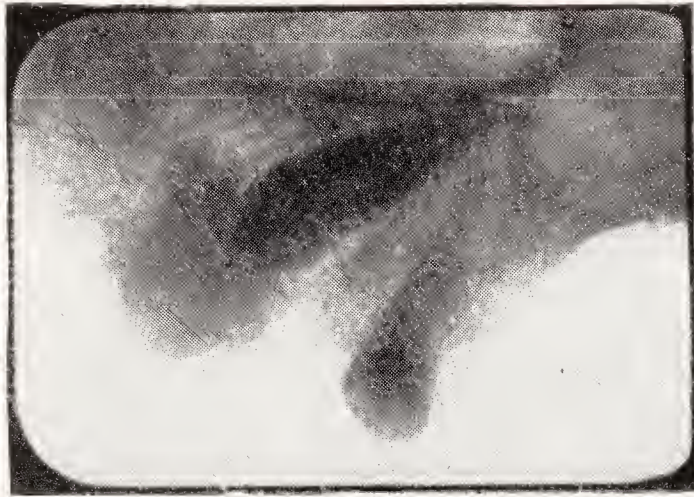


FIG. 432.—Radiograph of composite odontoma of maxilla. (Blum, Dent. Cosmos.)

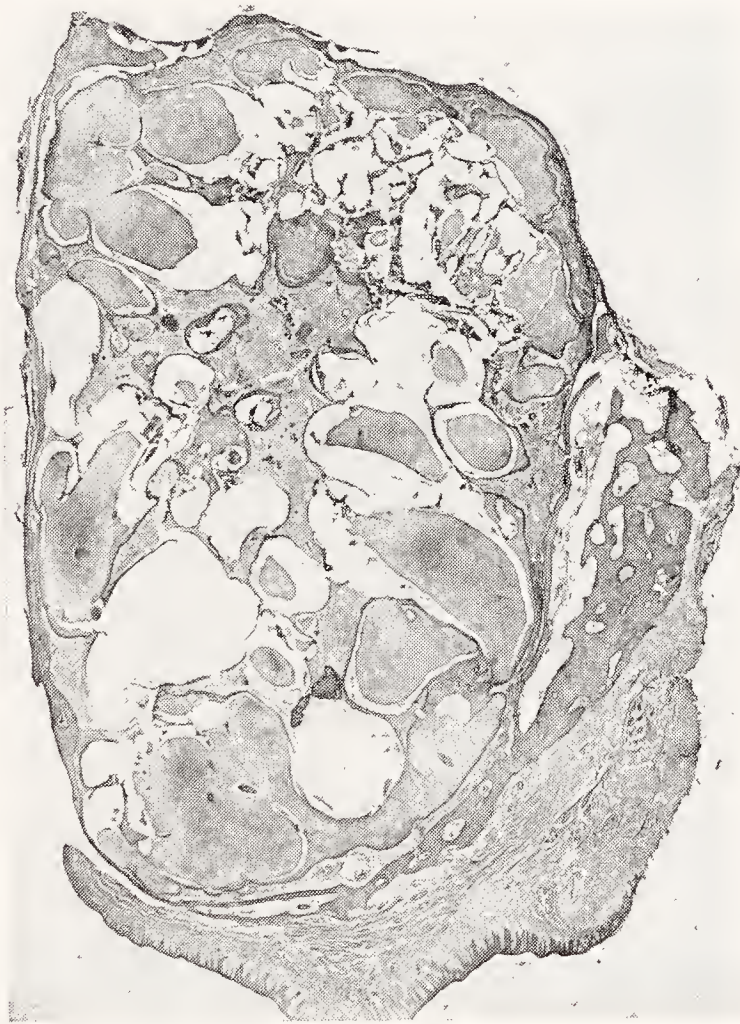


FIG. 433.—Photomicrograph of tumor shown in Fig. 432. Within the fibrous capsule several, more or less developed, small teeth are visible. (Enamel dissolved.) In the connective tissue, islands of ameloblasts and globules of calcium are present. (Blum, Dent. Cosmos.)

torted teeth. The odontoma may be completely covered with cementum, so that the enamel can be seen only after the tumor has been cut open.

Some odontomas consist entirely of irregular dentin and cementum- or bone-like tissue without enamel; these are called osteo-odontomas.

Usually it is not difficult to remove an odontoma surgically since



FIG. 434.—An odontoma and an impacted central incisor. (Cryer.)



FIG. 435.—Views of the impacted tooth and odontoma removed from the jaw illustrated in Figure 434. (Cryer.)

the tumor is not fused to the bone and can be lifted from its bed after the overlying tissues have been removed.

Ameloblastoma (Adamantinoma).—The ameloblastoma is a benign, soft, epithelial tumor originating from the odontogenic epithelium of the dental lamina and the enamel organ. Formerly the term “adamantinoma” was generally used to designate this

tumor; but, since literally translated this term means tumor of the enamel and the adamantinoma never contains enamel, the newer term "ameloblastoma" (tumor of the ameloblasts) should be given preference.

Ameloblastomas occur far more frequently in the mandible than in the maxillæ. Usually they begin to develop early in life and, as a rule, are discovered before the age of thirty years. These and other important facts were brought out by Robinson in a survey of 379 cases.

An ameloblastoma usually begins as a small, solid, fleshy tumor near the jaw surface. In its further growth it invades the jaw bone and then grows at the expense of the bone, in time causing extensive bone destruction and distention of the thinned outer bone shell. During their growth most ameloblastomas change from a solid to a cystic tumor. The cysts are separated by thin septa of bone, and, as a result, the radiographic picture of a typical cystic ameloblastoma shows multiple cystic spaces in the jaw bone, a condition known clinically as *multilocular cysts* (Fig. 436).

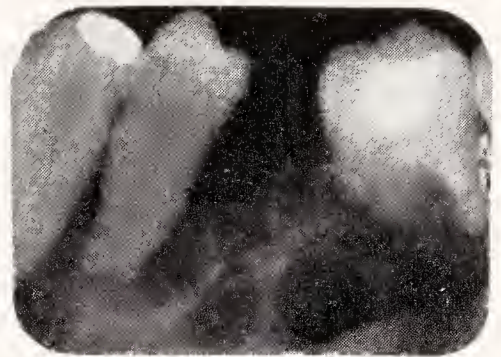


FIG. 436.—Cystic adamantinoma of the jaw bone. (Ennis.)

Several cases have been reported in literature in which an ameloblastoma developed from a dentigerous cyst. Apparently the cyst epithelium, which is derived from the enamel organ, may proliferate and assume the clinical and pathological characteristics of an ameloblastoma (Cahn).

The typical clinical history of an ameloblastoma is one of slow growth over a long period of time, sometimes as long as ten or twenty years. Frequently there is a history of repeated operations with incomplete removal of the tumor and recurrence. Eventually the jaw bone may become so extensively involved that it becomes necessary to resect a portion of it.

Although the ameloblastoma is classified as a benign tumor, malignant changes and formation of metastasis have been observed in atypical cases. But, generally, an ameloblastoma may be considered a benign growth with a tendency to recur.

The microscopic pathology of an ameloblastoma is very interesting. In its early development the tumor consists of a stroma of vascular, young connective tissue containing numerous strands and clusters of epithelium that bear a close resemblance to a dental lamina and dental anlagen in the early stages of tooth development.

Like the dental lamina, the epithelial tissue in the ameloblastoma is a continuation of the basal layer of the overlying jaw epithelium. Later, growth and differentiation take place within this epithelium, and areas of stellate reticulum appear. Toward the surface the epithelial cells are tall and cylindrical and resemble ameloblasts (Fig. 437). The connective tissue stroma of the tumor divides the epithelial part into small lobes. Up to this stage of development

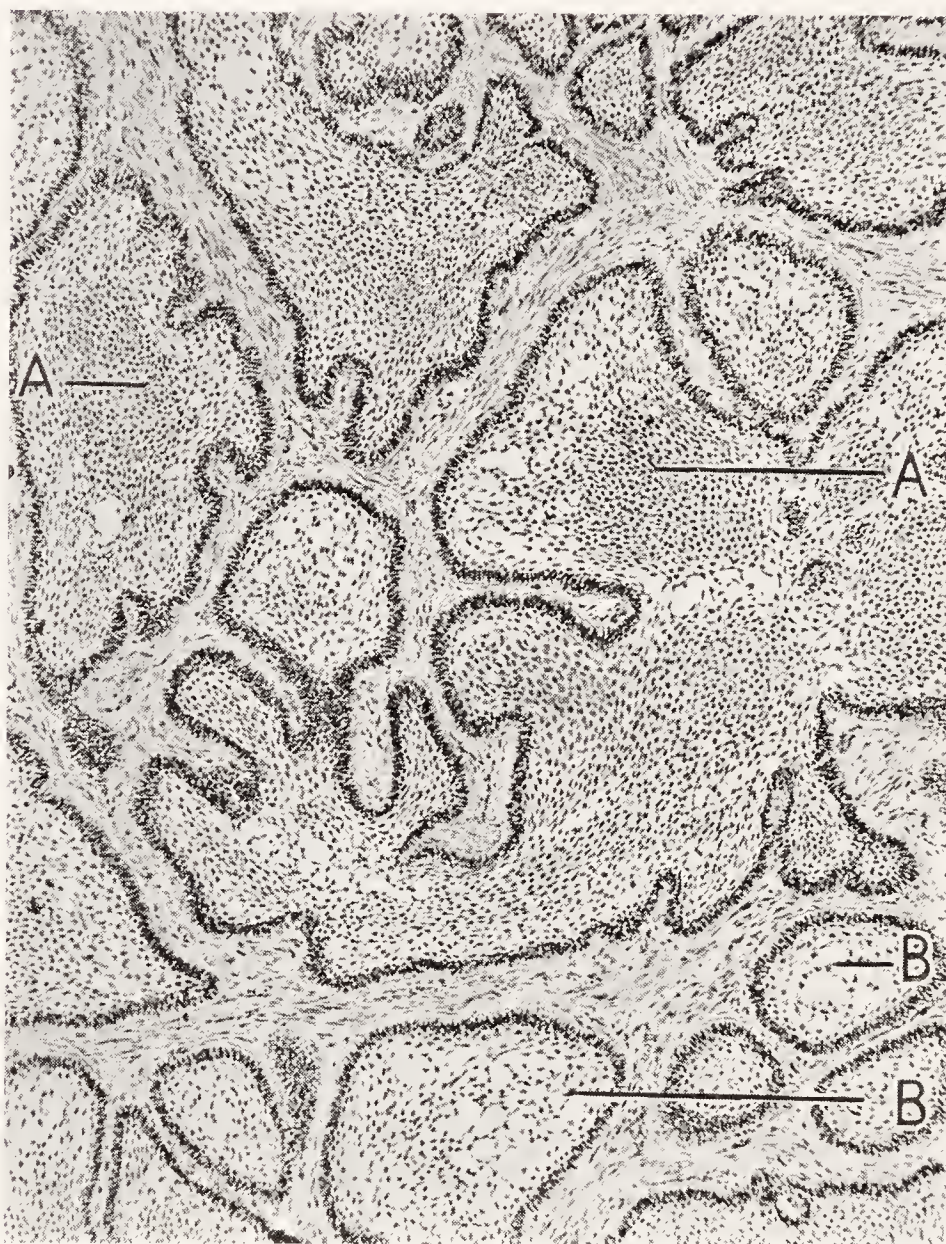


FIG. 437.—Ameloblastoma. The tumor consists of lobules that contain stellate reticulum and are lined with cylindrical cells. At *A*, the stellate reticulum is very dense. At *B*, a looser arrangement is shown. The lobules are surrounded by fibrous connective tissue. (Kronfeld, Jour. Am. Dent. Assn.)

the ameloblastoma imitates normal tooth development, but after this stage the parallelism ends. No enamel is formed; instead, cyst formation begins in the stellate reticulum, and gradually the entire tumor undergoes cystic degeneration. At first the cysts are microscopically small; however, they eventually become macroscopically visible and cause resorption of the surrounding bone; they lie in separate spaces or compartments in the bone, which cause the typical radiographic appearance of a cystic ameloblastoma.

TUMORS AND DERMOID CYSTS CONTAINING TEETH.

An interesting fact with which dentists are less familiar than general pathologists is that under pathological conditions teeth may be found in parts of the body other than the mouth and jaws. One location where teeth in tumors are occasionally observed is the brain; these tumors originate in the region of the pituitary gland. Pflüger and Schürmann reported two such tumors each of which contained numerous small teeth embedded in connective tissue. Even before death these teeth could be seen in radiographs of the head. Histologically, the teeth were rather well formed; they had short roots and the typical arrangement of enamel, dentin, and pulp in the crowns. Some of them were fastened to the bone tissue within the tumor. The tissue surrounding the teeth contained epithelial tissue that resembled dental lamina and early tooth germs.

The tooth-containing tumors of the pituitary gland are derived from epithelial remnants of the cord or tract of epithelium (hypophyseal duct) which early in fetal life extends from the pharynx toward the base of the brain and gives rise to the anterior lobe of the pituitary. This epithelium is split off from the oral epithelium at a time when the latter still has general odontogenic properties; in case of tumor formation and associated pathological cell stimulation and proliferation, the epithelium may become activated and cause tooth formation within the tumor.

Teeth are also frequently found in dermoid cysts of the ovary. A dermoid is a form of tumor known as a teratoma; the latter is defined as a tumor or tissue mass composed of structures derived from more than one germinal layer. A teratoma may be considered as an attempt to form a new individual within the body of another. The description of dermoid cysts of the ovary will help to make this clear.

An ovarian dermoid is a slow-growing, benign tumor, partly solid and partly cystic. The solid part consists of connective tissue and may contain cartilage, bone, muscle tissue, nerve tissue, and glandular structures. The cystic part is lined with skin (hence the name dermoid); from the skin, hairs project into the cyst lumen, which is filled with a fatty, tallow-like matter (Fig. 438). One out of every three or four ovarian dermoids contains teeth, which vary in number from one to fifteen, although two to five are most commonly observed. They resemble the teeth normally found in the mouth; molar-shaped crowns especially are often found. The roots are short and embedded in bony sockets located within the wall of the dermoid cyst. The crowns may protrude into the cyst lumen, or the entire

tooth may be buried in the solid portion of the dermoid. On a few occasions, erupted and unerupted teeth have been found in close relation in the same dermoid cyst, a condition comparable to that normally found in the teeth of the deciduous and permanent dentition (Kanamori).

A common observation is the occurrence of resorption in ovarian teeth. Sometimes the crown is resorbed, sometimes the root, often



FIG. 438.—Portion of a wall of an ovarian dermoid cyst. *a*, wall of the cyst; *b*, projecting portion made up of fatty and cutaneous tissue; *c*, hairs; *d*, teeth. (Burchard and Inglis.)

both. Bone and connective tissue usually occupy the resorbed areas. Defects of this sort may, upon superficial examination, resemble caries, and they have been responsible for reports of “caries” in such teeth; but here, as with fully embedded teeth, such a diagnosis is erroneous and is based upon failure to distinguish between caries and resorption (see p. 420).

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ADDENDA.

DIFFERENTIAL DIAGNOSIS OF TOOTHACHE.

- I. *Pain Originating from the Pulp*
 - (a) Hypersensitive dentin
 - (b) Hyperemia
 - (c) Pulpitis
- II. *Pain Originating from the Periodontal Tissues*
 - (a) Apical periodontitis
 - (b) Lateral abscess
- III. *Pain Originating from the Gingivæ*
 - (a) Papillitis
 - (b) Vincent's infection

The above classification gives a broad survey of the more common causes of pain in and around the teeth. The individual conditions have been discussed and their pathology illustrated in the body of the book; here, only the main symptoms will be enumerated, with special emphasis on the clinical differential diagnosis.

I. PAIN ORIGINATING FROM THE PULP.

(a) **Hypersensitive Dentin.**—To some degree at least, all exposed dentin is sensitive to touch, to sweet and sour foods, and to thermal changes. Usually this sensitiveness disappears in time because of dentin sclerosis and formation of secondary dentin. Occasionally, however, exposed dentin becomes more sensitive, especially in the cervical portion, until even a gentle touch on it, for example, with a tooth brush or a tooth pick, is very painful and forces the patient to consult a dentist.

The pathology of hypersensitive dentin is not fully understood. It appears to be partly a subjective symptom, depending upon individual sensitiveness and response to pain, and as such is difficult to evaluate. Treatment of the exposed area with caustics, such as silver nitrate, formalin, or zinc chloride, usually decreases the sensitiveness and brings relief from the symptoms.

(b) **Hyperemia of the Pulp.**—Hyperemia of the pulp is the result of pulp irritation, usually caused by the absence of a sufficiently thick protective layer of dentin covering the pulp. It occurs in teeth with

deep carious cavities, following tooth fracture, and in teeth with deep-seated metal fillings without proper insulation.

The most common exciting causes are cold and hot fluids or cold air. Ice cream, cold drinks, or hot soup causes a sudden twinge of pain. The main characteristic of this pain is that it disappears as fast as it comes: as soon as the cold or hot fluid has assumed mouth temperature, the pain subsides. The pain in pulp hyperemia is not spontaneous; so long as the mouth is closed and uniform temperature thus maintained, the tooth is comfortable.

A radiograph of such a tooth may reveal that a cavity or metal filling is in the vicinity of the pulp; the periodontal tissues appear normal.

Treatment consists of filling the tooth and providing adequate insulation with a material that is a poor conductor of thermal stimuli. If a metal filling is already present, it should be replaced by one with a thick insulating base. Sometimes the clinical symptoms of pulp hyperemia develop into those of pulpitis, and then the pulp can no longer be saved.

(c) **Pulpitis.**—Acute inflammation of the pulp is nearly always caused by caries and invasion of the pulp tissue by pathogenic microorganisms. The involvement of the pulp varies from a superficial inflammation of a pulp horn to a diffuse infiltration of the entire pulp, with or without the formation of a pulp abscess. The symptoms of pulpitis are typical, although their intensity varies widely. The pain may be spontaneous, or it may be elicited by thermal stimuli, especially by cold. The attacks of pain are prolonged, varying from a few minutes to several hours. The pain is of a sharp, lancinating type, often radiating into other teeth or into another division of the trifacial nerve of the affected side, so that the patient may be unable to localize the affected tooth. Often the pain is rather definitely assigned by the patient to another tooth in either the upper or lower arch. Frequently, spontaneous attacks of pain occur at night or upon lying down.

The intensity of the pain apparently depends upon several factors. One is the individual reaction to pain. Some people are extremely sensitive; others are surprisingly insensitive. Another factor is the location of the pulp involvement. If there is a large pulp exposure and the exudate from the inflamed pulp tissue can drain freely, the pain may remain very moderate. If, on the other hand, an acute inflammation or an abscess cavity develops in a pulp that is not freely exposed, pus and tissue fluid accumulate under pressure and, like every confined abscess, are likely to cause excruciating pain.

An objective examination usually reveals a deeply decayed tooth;

after the decay has been removed, the pulp may be seen as a red spot at the bottom of the cavity. The radiograph may show the extent of the decay and its proximity to the pulp. The apical region appears normal, except that occasionally in advanced stages, when there is incipient periodontitis, a slight widening of the apical periodontal space may be seen.

The outcome of acute pulpitis varies. If the patient seeks the services of a dentist, two things are possible: the dentist may extract the tooth, or he may anesthetize and remove the diseased pulp, and treat and fill the root canal. If no treatment is given, and the patient is able to endure the acute stage with the liberal use of anodynes, the inflammation may become chronic (chronic pulpitis) or the pulp tissue may die and become decomposed (necrosis and gangrene of the pulp).

In the advanced stages of pulpitis, when most of the pulp tissue is involved, the sensitiveness to cold decreases and is gradually replaced by sensitiveness to warmth. Later the tooth becomes sensitive to pressure and percussion. These changes indicate inflammation near the apical foramen and the beginning of involvement of the periodontal membrane.

There are only a few, mild clinical symptoms, or none at all, in chronic pulpitis. The tooth usually has a large cavity. The coronal portion of the pulp may be destroyed and the chronically inflamed tissue be confined to the root canal, or the pulp tissue may proliferate from the exposed pulp chamber, forming a pulp polyp. In either case there may be occasional, indefinite painful sensations or moderate pain when the pulp tissue is touched or injured during mastication or during examination. There is no, or very little, response to thermal changes. The radiograph usually shows a thickening of the apical periodontal membrane, indicating incipient inflammatory changes in the periodontium.

Chronic pulpitis, if untreated, leads eventually to destruction and gangrene of the pulp. With careful treatment, however, the pulp can be removed and the canal filled, and thus the tooth can usually be preserved.

II. PAIN ORIGINATING FROM THE PERIODONTAL TISSUES.

(a) **Apical Periodontitis.**—Acute periodontitis or pericementitis is an inflammation of the apical periodontium, usually caused by death of the pulp and an invasion of pathogenic microorganisms. The following clinical symptoms are noticeable: A tooth with or without a root canal filling is infected and pulpless. It is not sen-

sitive to cold, sometimes slightly sensitive to warmth, but always sensitive to percussion (tapping) and pressure. There are other symptoms that are not always found, but usually at least one or two of the following are also present: The involved tooth is slightly loosened. The patient feels that the tooth is raised in its socket. The radiograph usually shows bone destruction around the apex.

The pain in acute periodontitis is of a continuous, dull, throbbing nature, localized in the jaw bone surrounding the affected tooth. Often in the early stages the patient observes that firm pressure upon the tooth brings temporary relief. Later, pressure becomes painful, although momentary relief occurs if pressure is released suddenly.

In the early stages of acute periodontitis the treatment consists of opening the root canal and establishing drainage through it. If this does not give relief, or if the crown is badly destroyed, the tooth should be extracted.

The acute periodontitis may subside spontaneously after a few days, or the inflammatory process may spread and an acute dento-alveolar abscess may develop. In this condition the patient complains of dull pain and a feeling of heat, fullness, and tension in the affected region. The soft tissues overlying the diseased tooth may be swollen (cellulitis); finally pus makes its way to the surface, either through the alveolar process or alongside the tooth, through the socket.

The acute dento-alveolar abscess is accompanied by fever, general ill feeling, foul odor from the mouth, and swelling and soreness of the regional lymph nodes.

The treatment is directed mainly toward evacuation of the pus. Sometimes lancing of a subperiosteal abscess, sometimes extraction of the tooth will provide drainage and relieve the pain.

There is rarely ever pain in chronic dento-alveolar abscess, granuloma, or radicular cyst, except when an acute flare-up causes an acute dento-alveolar abscess. The diagnosis of chronic periodontitis is made from the presence of a pulpless tooth and from the radiographic evidence of bone destruction at the apex. The treatment consists, in favorable, selected cases, of root canal therapy; otherwise extraction of the involved tooth is indicated.

(b) **Lateral Abscess.**—A lateral abscess is an acute inflammatory condition and pus formation in a deep pyorrhea pocket. The symptoms resemble those of an acute dento-alveolar abscess, except that as a rule they are not so severe. The pain is dull and throbbing; the gingivæ around the affected tooth are swollen and puffy. A red wheal develops where the abscess points. Frequently pus can be

seen exuding from a deep pocket alongside the tooth. Usually the differential diagnosis between a lateral abscess and an apical abscess is not difficult: With a lateral abscess the tooth ordinarily has a vital pulp, and the apex is radiographically normal. The presence of a deep paradental pocket can be demonstrated with the aid of an explorer.

The treatment consists of lancing the abscess. Extraction of the tooth is usually indicated.

III. PAIN ORIGINATING FROM THE GINGIVÆ.

(a) **Papillitis.**—Occasionally acute inflammation of an interdental papilla causes considerable pain, which may resemble pulpitic pain. The cause of acute papillitis is usually a trauma, such as food packing against the papilla because of lack of contact point, foreign bodies wedged into the interdental papilla, such as a tooth brush bristle or a piece of a tooth pick, or repeated injuries caused by abuse of the tooth pick or dental floss.

By a careful examination of the mouth it is usually not difficult to find the inflamed, painful papilla. The cause of the inflammation can then be removed, a mild antiseptic applied, and the papilla heals in a few days.

(b) **Vincent's Infection.**—Acute Vincent's infection may cause considerable pain and sometimes makes it almost impossible for the patient to sleep or eat. The differential diagnosis is not difficult, since Vincent's infection in the acute stage is easily recognized. The clinical symptoms have been described in detail in Chapter XX. Care must be taken not to overlook teeth with pulpitis in the mouth of a patient with Vincent's infection; one condition by no means excludes the other, and failure to recognize and treat a pulpitic tooth in a mouth with Vincent's infection means that the patient will not be relieved of pain.

DIFFERENTIAL DIAGNOSIS OF CYSTS OF THE ORAL CAVITY.

I. *Cysts of Dental Origin*

- (a) Radicular or root cyst
- (b) Follicular or dentigerous cyst

II. *Retentive Cysts of Oral Glands*

- (a) Mucous cyst
- (b) Retentive cyst of sublingual gland (ranula)

III. *Cystic Tumors*

- (a) Cystic ameloblastoma or multilocular cyst

IV. *Cysts of Developmental Origin*

- (a) Median anterior maxillary cyst
- (b) Dermoid cyst

V. *Bone Cysts*

General definition of cyst: A cyst is a pathological cavity, lined with epithelium and containing a fluid or semifluid.

I. CYSTS OF DENTAL ORIGIN.

(a) **Radicular Cyst, Root Cyst, or Root End Cyst.**—A radicular cyst is always associated with an infected pulpless tooth. In the early stages a radicular cyst may be symptomless; later there may be a slowly growing, painless swelling of the jaw bone. The radiograph reveals an area of bone destruction into which the apex of an infected pulpless tooth protrudes; with a strong injection needle the cyst wall can be perforated and the cyst fluid aspirated. The latter is yellowish and ropy; it may contain bacteria of moderate virulence or it may be sterile. After the involved tooth is extracted, the cyst often persists and continues to grow slowly. The symptoms of an acute infection of a radicular cyst are the same as those of an acute dento-alveolar abscess: pain, cellulitis, and discharge of pus.

The epithelial lining of a radicular cyst consists of stratified squamous epithelium derived from the epithelial rests of Malassez of the periodontal membrane. The fibrous cyst sac originates from the connective tissue of the apical periodontal membrane and from the periosteum of the alveolar process.

There are two possible treatments: (1) to enucleate the entire cyst sac, or (2) to open the cyst and to maintain the opening until the bone has filled in from beneath. The tooth from which the cyst originated usually cannot be saved.

(b) **Follicular or Dentigerous Cyst.**—A follicular or dentigerous cyst is the result of cystic degeneration of the tooth follicle of an unerupted tooth. The cause for this condition is unknown. Clinically, a dentigerous cyst appears as a slowly growing, painless, non-inflammatory swelling of the jaw bone. The radiograph shows an area of bone destruction surrounding the crown of an unerupted tooth; the crown projects into the cyst lumen. Upon aspiration with a needle, a clear, yellowish, sterile fluid can be withdrawn.

The epithelial lining of a dentigerous cyst is derived from the enamel epithelium of the tooth germ; the connective tissue surround-

ing the cyst, the cyst sac, originates from the connective tissue of the tooth follicle. A dentigerous or follicular cyst is essentially a pathological accumulation of fluid between the enamel of an unerupted tooth and the surrounding soft tissue; as the cyst grows, the tissues investing the crown are distended by the cyst fluid but remain attached to the tooth along the cemento-enamel junction.

The treatment consists of opening the cyst to relieve the pressure of the cyst fluid. Sometimes the tooth later erupts and assumes a normal position in the arch. If, however, the tooth in the cyst is so situated that eruption appears impossible, it should be removed surgically.

II. RETENTIVE CYSTS OF ORAL GLANDS.

(a) **Mucous Cyst.**—A mucous cyst is a small, cystic sac that develops in the mucous membrane of the lips or cheek. It is the result of an obstruction of the duct of one of the many, small mucous glands of the oral mucosa. Mucous cysts are most commonly found in areas where the mucosa is subjected to repeated minor injuries, such as the inside of the lower lip. They grow until they form a bluish sac of several millimeters in diameter. Then they usually rupture and may temporarily disappear; somewhat later, however, they may begin to fill again.

The treatment consists of surgical excision of the cyst sac.

(b) **Ranula.**—A ranula (the word means “small frog”) is a retentive cyst of the sublingual gland. It appears as a bluish-white, transparent sac on the floor of the mouth, under the tip of the tongue, where the opening of the sublingual duct is located. A ranula usually grows slowly and painlessly until it reaches the size of a large cherry. It may be caused by anything that blocks the free flow of saliva from the sublingual duct, such as salivary stones, inflammation of the floor of the mouth, or injuries.

The treatment is surgical; it consists of establishing the free flow of saliva from the sublingual gland by incision and removal of the obstacle.

III. CYSTIC TUMORS.

(a) **Cystic Ameloblastoma or Multilocular Cyst.**—An ameloblastoma or adamantinoma is a soft, benign epithelial tumor of the jaws. It consists of multiple epithelial strands embedded into a connective tissue stroma. The epithelial portion of the tumor is derived from the dental lamina and from the early tooth anlage. Thus, an ameloblastoma may be considered a pathological overgrowth (tumor) of a tooth in its earliest stages of development.

Most ameloblastomas are found in the mandible. In the later stages the epithelium of the tumor undergoes extensive cystic degeneration and forms multiple cysts which, in advanced cases, may destroy most of the jaw bone (multilocular cysts). The radiograph reveals the multiple cysts and usually shows bony septa separating the individual cyst cavities. The cysts contain a clear, sterile fluid; their epithelial lining is derived from the tumor epithelium, their surrounding connective tissue from the stroma.

The treatment consists of surgical elimination of the tumor; sometimes resection of the jaw becomes necessary.

IV. CYSTS OF DEVELOPMENTAL ORIGIN.

(a) **Median Anterior Maxillary Cysts.**—A median anterior maxillary cyst is found in the midline of the upper jaw, between and above the upper central incisors. It occupies the lower portion of the anterior palatine canal. There are no clinical symptoms, and the condition is usually discovered accidentally by radiographic examination.

A median maxillary anterior cyst is the result of cystic degeneration of epithelial tissue that has become embedded in the anterior portion of the maxilla during early fetal life when the hard palate was being formed. The cause for the development of this type of cyst is unknown.

As a rule, no treatment is indicated, since these cysts cause no harm and grow very slowly. Only rarely do they become large enough to interfere with the adjacent structures.

(b) **Dermoid Cyst.**—A dermoid cyst is a cyst of developmental origin, containing skin, hair, nails, teeth, and various other structures of the body. It grows slowly and painlessly; it reaches the size of a fist or even larger and contains a fatty, tallow-like material. Most dermoid cysts are found in the ovaries or testicles; in rare instances, however, they occur in the floor of the mouth and require surgical removal.

V. BONE CYSTS.

Occasionally the radiograph reveals cysts in the jaw bone that do not fall into any of the above-mentioned classes. Waldron has suggested the term "indeterminate type cyst" for them. They appear as small, round, sharply outlined defects in the bone. Their etiology is unknown. Often they are not cysts in the true sense of the word, as they may lack an epithelial lining and be merely a bone cavity containing a hemorrhagic fluid. The latter type of bone cyst may be related to certain rare bone diseases, such as osteitis fibrosa cystica.

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